THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
ON DISEASES OF THE

EAR-NOSE-THROAT

VOLUME LX — JANUARY-DECEMBER, 1950.

FOUNDED IN 1896 BY
DR. M. A. GOLDSTEIN, ST. LOUIS
DR. THEO. E. WALSH, ST. LOUIS
EDITOR

PUBLISHED BY THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY - ST. LOUIS (10), MO., U.S.A.

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THE

LARYNGOSCOPE.

VOL. LX JANUARY, 1950. No. 1

HISTIOCYTIC GRANULOMA OF SKULL.*

(A Triphasic Clinicopathologic Syndrome Previously Termed Letterer-Siwe's Disease, Hand-Schüller-Christian's Disease, and Eosinophilic Granuloma.)

Report of 18 Cases.

VICTOR GOODHILL, M.D.,
Los Angeles, Calif.

INTRODUCTION.

Histiocytic granuloma of the skull is a clinicopathologic entity which may appear in an acute, usually fatal form known as Letterer-Siwe's disease, a subacute serious stage known as Hand-Schüller-Christian's disease, and a chronic benign state known as eosinophilic granuloma.

Confusion reigned for several years as pathologists and clinicians diligently attempted to classify each one of the above phases into a separate distinct disease category.

Evidence will be presented to clarify the relationship between these three "diseases" and to demonstrate the fact that they are truly different phases of one single disease process, histiocytic granuloma.

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*Submitted as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1948.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 1, 1949.

Of great interest to the otolaryngologist is this disease with its frequent manifestations of otorrhea, temporal bone destruction, multiple skull erosions, proptosis, and diabetes insipidus.

The important contributions to the literature are summarized and 18 new cases are reported, illustrating the three phases of the disease.

HISTORICAL ASPECTS AND DEVELOPMENT OF PREVIOUS CONCEPTS.

1. Development of the Concept of Hand-Schüller-Christian's Disease.

In 1893, Alfred Hand published the report of a case showing a clinical triad of calvarial osseous defects, exophthalmos and polyuria in a three-year-old child. Because of the caseous-like appearance of the granulation process, Hand attributed the disease to tuberculosis and entitled his publication, "Polyuria and Tuberculosis." He felt that tuberculous deposits in the calvarium and skull base were associated with symptoms of polyuria.

In 1915, Arthur Schüller presented his concept of the disease. He described three cases, in each of which there were irregular defects in the membranous bones. His first case was that of a 16-year-old boy with infantilism, exophthalmos and the adiposogenital syndrome; the second case was that of a female, age four, with stunted growth, exophthalmos and diabetes insipidus; and the third case was an adult with skull lesions and an infiltration of the skin with yellow nodules. He did not make any suggestions regarding the etiology or pathology of the disease, but he simply pointed out the common radiological fact of irregular skull defects in all three cases; however, in 1926, Schuller felt that the disease was of pituitary origin and proposed the name "dysostosis hypophysaria."

In 1919, Henry Christian published an article entitled, "Defects in the Membranous Bones, Exophthalmos and Diabetes Insipidus." He reported the case of a five-year-old girl with the triad mentioned and suggested that the diabetes insipidus was due to a disturbance of pituitary function. Then he pointed out that the polyuria was controlled by pituitrin intramuscularly.

In 1921, Hand reported another case in which he described a three-year-old child with exophthalmos, polyuria and an area of softening in the center of the right parietal bone. In this case again, as in 1893, Hand attributed the disease to tuberculosis, but he stated: "The primary process might be neoplastic, benign and myxomatous in character, affecting, for some unknown reason, the membranous bones and producing exophthalmos and polyuria secondarily by pressure." The pathological report mentioned the fact that there was a defect in the center of the right parietal bone occupied by a soft yellow material resembling the cavitation seen in tuberculosis.

In none of the publications by the above three authors, whose names have been linked to create a new disease entity (Hand-Schüller-Christian's disease), was there any distinct pathological description which would in any way fit the common present conception of the disease. None of the three men succeeded in actually presenting the features of a granulomatous lesion of lipoid character, perhaps of histiocytic origin.

In 1925, Thompson, Keegan and Dunn presented a case report of a nine-year-old boy with the classical triad: exophthalmos, calvarial defects and polyuria. In describing the autopsy findings, it is stated: "In the center of the largest membranous area, that is, the calvarial defect, there was yellowish fibrous tissue; and the inner surface of the dura mater was mottled by a yellowish tissue. Microscopic examination of the area showed a lining layer of large oval cells with sharp borders and clear, slightly granular cytoplasm and small, compact, centrally placed nuclei. The cytoplasm contained a variable amount of lipoid material in finely divided form." This was the first time in the history of the disease in which the existence of lipoid in the lesion was described.

Suffice it to say that the concept of the triad known today as Hand-Schüller-Christian's disease stems from the original reports of these three pioneer investigators, whose fine observations and keen clinical judgment justified the attachment of their names to this newly recognized entity, in spite of the fact that they did not describe the basic histopathologic features of the disease.

It remained for Dr. R. S. Rowland, in 1928, to correlate the various findings and to clarify the histopathology of the syndrome for the first time. He concluded that this syndrome of Hand, Schuller and Christian consisted of a form of xanthoma in which portions of the reticuloendothelial system became the site of excessive lipoid storage with resulting hyperplasia of lipoid-containing cells. He stated, "One is not dealing with true neoplasm but with hyperplastic new formation, *i.e.*, lipoid storage tumors. Formation of these nodules is a compensatory act on the part of the body in its attempt to rid the blood of an excess of lipoid which cannot be properly excreted." It was probably Rowland who, for the first time, applied the name xanthomatosis to the Hand-Schüller-Christian syndrome and who first attached the disease to the reticuloendothelial system insofar as histopathology was concerned.

When one considers the vast group of confusing diseases variously termed lipoidoses, one is confronted by a confusingly large number of diseases to which men's names have been attached. In discussing the lipoidoses, in some instances chemical methods have been used to outline distinctions. Thus, the clinical condition known as Niemann-Pick's disease is considered to be that entity in which a lipoid of the lecithin or phosphatide type is deposited in the lymphovascular and hematopoietic tissue of the liver, spleen and bone marrow. Another disease is Gaucher's, in which the lipoid material is a cerebroside. Xanthomatosis is a lipoidosis in which the lipoid material is cholesterol. Tay-Sach's disease is a condition in which the essential feature is a phosphatide lecithin lipoid accumulation in the tissues of the central nervous system. A great deal of quarreling and argument ensued in the literature for years regarding the differential diagnosis of these various lipoid diseases; and even today there is no unanimity among pathologists or among biochemists regarding the fine differentiation that has been accorded these various diseases.

Certain pseudoscientific criteria for the differentiation of these diseases have been set up with a resultant increase in confusion among clinicians and pathologists. Ludwig Pick, who has written a great deal on these diseases, stated in 1933 in a discussion of Niemann-Pick's disease and other so-called xanthomatoses that Hand-Schüller-Christian's disease was an essential xanthomatosis. He stated, "It occurs predominantly in Jews and as far as we know in the male sex." He continues, "The lipoid deposition here consists chiefly of cholesterin fatty acid esters. The accumulation of large storage cells is secondary to a very characteristic granulation tissue which has an outspoken tendency to conversion into fibrous scars." In further discussing the disease, he goes on to give the clinical aspects of the triad as described above; and in addition he states that there are growth disturbances, dystrophia adiposogenitalis; yellow-brown skin discoloration; dyspnea; cyanosis; enlargement of the liver, spleen and lymph nodes. He stated that the foam cells eventually cicatrized, especially in the liver. He mentioned the observation that there is a complete replacement of bone marrow by masses of foam cells with subsequent formations for granulomatous tissue, which in turn produces the bony defect noted Roentgenologically.

It is of passing interest that Pick demonstrates his ignorance of modern anthropology by making the bald statement that Hand-Schüller-Christian's disease occurs predominantly in the Jewish "race." (Present-day scientific anthropology denies the authenticity of a so-called Jewish "race.") In the present report of 18 cases, there are none in Jewish patients.

He also stated that it occurred only in the male sex; and in this series of 18 cases to be presented, there were seven females and 11 males.

Dr. John Fraser published a masterful analysis of the Hand - Schuller - Christian entity entitled, "Skeletal Lipoid Granulomatosis." He described it as a disease of the so-called reticuloendothelial system in which there occurred the formation of a lipoid granuloma. He went into great detail to describe the reticuloendothelial system in an attempt to prove the histological rationale for the depostion of lipoids into these various cellular areas and also in an attempt to explain formation of some of the unusual histological structures such as the foam cells and giant cells seen in this disease. In describing the reticuloendothelial system, Fraser repeated the old concept that the system included the endothelium lining blood and lymph vessels. He then went on to mention the usual reticulum cells, the histiocytes and the monocytes in the blood stream. (He ignored the Maximow concept which is today more universally accepted; namely, that the reticuloendothelial system is a misnomer inasmuch as the reticulum cell, or histiocyte, is not related morphologically or developmentally with the endothelial cell lining of blood and lymph vessels. Maximow and Bloom stated that the most appropriate name for this type of cell form was the "histiocyte.")

Fraser, in pursuing his original thesis that Hand-Schüller-Christian's disease was a skeletal lipoid granulomatosis in which cholesterol was being absorbed and stored by the reticuloendothelium or histiocytic system, felt that hypercholesteremia was a very important factor in the production of this disease. He felt that an excess of lipoid in the circulating blood was due to a failure of the liver and lung tissue to maintain the body lipoid at a normal level.

Accordingly, it was his thesis that the reticuloendothelial tissue attempted to eliminate the excess by a process of absorption and deposit. This deposition of lipoid, according to Fraser, produces a considerable cell reaction in the reticular condition concerned; and it is this cellular reaction which produces the characteristic pathology of the lesion. In speculating upon the mechanism of the granulomatous formation, Fraser stated as follows: "The formation of the granulation elements is associated with certain untoward results. Its ability to infiltrate is insignificant, but it spreads with relative ease into parts occupied by areolar tissue and such fat-containing areas as the orbit and the perivascular spaces. What appears to be an infiltration of an organ is an extension along perivascular channels, and it is evident that fibrous tissue presents an impenetrable barrier to its invasion. Its progress in bone is marked by a progressive and extensive decalcification of trabecular and later compact bone with the result that large portions of the calvaria, clavicles or mandible—all more or less membrane bones—may disappear; and from what we find, it seems that the process of bone removal is one of osteolysis or phagocytosis initiated and accomplished by the larger type of multinuclear giant cell which is universally found at the periphery of the lesion. It is significant that, although large areas of bone may be involved and the skull wall reduced to a condition which has been described as gelatinous, the underlying dura and the overlying scalp often remain unaffected. The deposits in the basal subdural area are rare except for the space which lies around the hypophysis. This area is so frequently affected that it may be regarded as a site of election, and in some instances the sella turcica is filled with a putty-like mass with the result that there is displacement of the hypophysis and traction or pressure on its stalk. In a few instances, infiltration of the hypophysis and of the hypothalmic area has been described, but such is rare. It is a remarkable feature that, though there may be pronounced accumulation in the sella turcica, there is no invasion of the basisphenoid or of the clinoid processes.* From the hypophyseal fossa, the granulation tissue extends forward around the carotid vessels and then to the tissue around the optic nerve, until it finds itself in the orbit where factors of space and fat content permit such accumulation of the deposits and the eyeball is displaced. The effect of the disturbance in regions other than bone is a displacement process. Surrounding elements are pushed aside, depressed and atrophied. Tissue spaces are filled and distorted; and as far as can be observed, it is only in relation to bone that one witnesses the phagocytosis which so intimately affects the skeletal outline. The summary of the pathology is, briefly, that in certain tissue areas there is a formation of granulation tissue of distinctive cytology, the result of reactive changes in the reticuloendothelial tissue, the process being a reaction to a cholesterol excess in the blood and body fluids."

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*This has not been true in my case reports.

Fraser, in his clinical discussion, tended to oversimplify the disease, and in his anxiety to define clearly the limits of the disease as a lipoid granuloma, he perhaps was a little too enthusiastic in some of his conclusions. He states, "The blood readings show an excess of cholesterol, of total acids and of lecithin. The characteristic histological picture is a proliferation of the reticuloendothelial cell groups, illustrating the presence of large numbers of phagocytic cells, multinucleated giant cells and large lipoid-containing or foam cells."

John Fraser's contribution to our conception of this disease was monumental.

Following the clear-cut descriptions of the above-mentioned authors, numerous publications quickly began to appear in the literature, particularly in the pathologic, radiologic and otolaryngologic fields.

In 1938, Shea reported one case of xanthomatosis which responded to X-ray therapy.

In 1932, M. C. Sosman discussed the subject of xanthomatosis and added two new cases.

In 1935, Lederer, Poncher and Fabricant added one case report to the literature. It was an instance of temporal bone involvement without otorrhea where the only findings were those of a granuloma in the external auditory canal and calvarial involvement. The process was favorably reversed by X-ray therapy.

In 1936, J. G. Druss reported a case of temporal bone involvement by Hand-Schüller-Christian's disease in a 48-year-old man. The patient had no otorrhea but had diabetes insipidus. The temporal bone section showed involvement, but it was discovered accidentally on routine temporal bone studies. There were no clinical manifestations during life.

In 1937, V. V. Wood presented a case of bilateral mastoid involvement by xanthomatosis.

In 1940, D. S. Kellogg presented one case report of a parietal lesion that was palpable physically and seen on X-ray. There was no temporal bone involvement. There was no biopsy. A diagnosis of xanthomatosis was made purely on the response of the parietal lesion to X-ray therapy.

In 1946, M. A. Glatt presented a patient with otorrhea who had other evidence of Hand-Schüller-Christian's disease.

In 1940, Rosenwasser reported an interesting case of a patient who had been diagnosed as having a temporal bone neoplasm called carcinoma simplex. The tissue was re-examined four years later and rediagnosed as xanthoma. The patient responded well to X-ray therapy.

In December, 1938, R. L. J. Kennedy reported eight cases of Hand-Schüller-Christian's disease from the Mayo Clinic. All of these patients had demonstrable skull defects; none of them had changes chemically in the blood lipoids. All of the patients were either improved or entirely well when last heard from. The treatment of choice in all these cases was the local application of X-ray or radium in small suberythema doses. Gradually, the author reports, the defects in bone were replaced by normal bone which obliterated in most instances all evidence of the original lesion.

In 1939, Cooper reported the case of a 13-year-old child with diabetes insipidus and a postauricular abscess which responded to surgical drainage and postoperative X-ray therapy.

In 1940, Hamblen, Arena and Cuyler reported one case of Hand-Schüller-Christian's disease in which urinary androgens were studied. These androgen excretions amounted to 10 times the normal value. It was felt that this was due to the general disturbance of lipid metabolism and not due to any hypercortical or hypergonadal activity.

Many of the specific manifestations of Hand-Schüller-Christian's disease have been subjected to careful clinico-pathological study. For example, ocular symptoms were very thoroughly analyzed in 1933 by Sanguinetti, who stated that the pathogenesis of the exophthalmos is based upon deformation of the orbit due to softening and rarefaction of the bony walls. The bony floor of the cranium may be converted into a membrane which is depressed by the weight of the overlying cerebral mass and results in flattening of the orbital cavity, compression of the optic nerve and partial atrophy. Pressure also displaces the eyeball and results in exophthalmos.

Carl Behr, in 1936, in another study of the ophthalmologic changes in Hand-Schüller-Christian's disease, tried to subdivide the various lipoidoses insofar as their ocular manifestations are concerned. He reported a monosymptomatic circumscribed instance in which an orbital involvement followed trauma and in which the patient recovered completely following surgical extirpation of the mass. He was able to demonstrate a hypercholesteremia in this patient and noted the interesting observation that here was a man of 69 years who, in spite of hypercholesteremia and Hand-Schüller-Christian's disease, did not have any evidence of generalized arteriosclerosis.

Lane and Smith, in the discussion of the cutaneous manifestations of chronic idiopathic lipoidosis, or Hand-Schüller-Christian's disease, report four cases. According to their studies, cutaneous manifestation are present in only one-third of the cases of Hand-Schüller-Christian's disease. According to these authors, there are two common types of eruption in this disease: the petechial and the diffuse papular. It seems that the diffuse petechial hemorrhagic eruption occurs in severe and acute cases; whereas with protracted disease, papular or nodular lesions may appear. The papules are at first bright red but later assume a yellow or brown color from the xanthomatous deposits in the skin. The petechial exanthem would indicate that in the more fulminating types there is damage to the capillaries with hemorrhages into the skin. These authors feel that the skin manifestations should be studied more closely inasmuch as they may very well be diagnostic in the absence of the complete triad of exophthalmos, polyuria and calvarial lesions.

Slesinger, in 1936, reported a fatal case in a three-year-old child whose chief complaint on admission was a swelling in the postauricular region. There was no otorrhea. There were no other significant findings. A section was reported from the biopsy of the postauricular mass as showing a peculiar form of granulation tissue with many polyhedral cells that had the appearance of being histiocytes. Further biopsy was reported as showing essentially the findings of lipoid histiocytosis. The child was given deep Roentgen ray therapy, but there did not seem to be any response to treatment; however, a direct implantation of radium into the postauricular lesion produced complete healing within three weeks. Within several months, new lesions appeared in lymph nodes, in the left parietal region and in the right knee; finally, polyuria and polydipsia appeared. At no time was there any exophthalmos.

Wynkoop and Hadley, in 1938, reported a case of a child with numerous skeletal lesions but no exophthalmos or diabetes insipidus. He apparently made a complete recovery following diffuse X-ray therapy.

Rudolph Klotz, in 1940, describes one case, termed by him as Christian's syndrome rather than Hand-Schüller-Christian's disease because of the fact that only a geographic skull was found. This patient, in addition to a parietal lesion, also had a splenic tumor and a disturbance of liver function. The author considered that this was a case of monosymptomatic "Christian's" disease.

Robert Strong, in 1936, reviewed the literature and stated, "Xanthomatosis may be regarded as being a clinical disease due to a disturbed lipoid metabolism which is not necessarily related to the general metabolism of fats, but is, in a large measure, influenced by the part played by the reticuloendothelial system.

In 1936, Dauksys reviewed the literature to date and reported one case.

In 1935, Teperson reported the case of a 16-month-old child with proptosis, later followed by other cranial defects; no diabetes insipidus appeared in this child. X-ray therapy was apparently successfully used.

II. Development of the Concept of Letterer-Siwe's Disease.

In 1936, Abt and Denenholz published a paper in which they proposed the name "Letterer-Siwe's disease" for a clinical entity of "splenohepatomegaly associated with widespread hyperplasia of the nonlipoid storing macrophages." They gave credit to E. Letterer and S. A. Siwe for earlier descriptions of clinical characteristics and pathological definitions.

In a lucid description of this entity, Tracy B. Mallory states, "Typical Letterer-Siwe's disease rarely occurs beyond two years of age and runs a rapidly and, it is at present believed, inevitably fatal course. It is characterized clinically by fever, skin rash, rapidly progressive anemia and purpura, and histologically by marked proliferation of the so-called 'reticuloendothelial' (monocytic and clasmatocytic) cells in many parts of the body, particularly the skin, lymph nodes and the spleen."

Letterer termed this "disease" aleukemic reticulosis. Siwe called it diffuse reticuloendotheliosis. Foot and Olcott suggested the term nonlipoid histiocytosis.

III. Development of the Concept of Eosinophilic Granuloma.

In 1940, Lichtenstein and Jaffe reported a case in which they stated as follows: "We propose that this lesion be designated eosinophilic granuloma of bone. It presents itself as a rather well localized single lesion starting in the medullary cavity and tending to erode, expand and perforate the cortex in the bone site affected. In microscopic appearance there are aggregates of large phagocytic cells with conspicuous collections of eosinophilic leucocytes interspersed." They suggested that this lesion may be a virus granuloma.

In the same year, Otani and Ehrlich described four cases of solitary bone lesions which seemed to be benign. They did not find any lipophages; therefore, they felt that they were not dealing with a "lipogranuloma of Hand-Schüller-Christian's. Because of the absence of doubly refractile fat, they ruled out the latter disease. Their histological studies showed a predominance of histiocytes and eosinophiles. They suggested that these granulomata are of traumatic origin.

In 1944, Jaffe and Lichtenstein made another contribution to the literature in which they reported several new cases and advanced the concept of nosologic independence of eosinophilic granuloma as a pathological and clinical entity. They made a very strong plea for the dissociation of this disease from the Hand-Schüller-Christian group. In discussing the histopathological picture, they stated as follows: "When an affected region of bone, not fractured, in which the lesion is in an early phase is entered, it is likely to be found more or less hemorrhagic and cystic and to show a relatively small amount of soft, brownish granulation tissue which may be streaked with yellow. Indeed, much of this granulation tissue may be found necrotic. Characteristically, the non-necrotic tissue represents, on microscopic examination, conspicuous sheet-like collections of large phagocytic cells of the nature of histiocytes, interspersed among which are more or less conspicuous numbers of eosinophilic cells and especially of eosinophilic leucocytes; furthermore, one finds actively phagocytic, multinuclear, giant cells especially in the vicinity of fields of hemorrhage and necrosis. *It is the histiocytes that constitute the basic component of the lesion.** Most of them contain a

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*Italics are the present writer's.

5. The condition is practically limited to children and young adults, especially males.

6. Surgical treatment is the most desirable procedure, although the disease is amenable to X-ray therapy. Spontaneous healing, however, is known to occur, unassisted by any other therapy.

7. Although not proved, it is thought to be associated in some manner as a stage of Schüller-Christian's disease and Letterer-Siwe's disease, representing a different expression of the same basic lipoid disorder.

8. The cause is obscure, but is most probably the result of some infectious agent. The part played by trauma, as stressed by several authors, does not seem to be substantiated. Some obscure virus infection has also been postulated. As yet, the pathogenesis has not been established.

9. Recognition of the benignity of the lesion will result in a more conservative therapy in certain obscure bone lesions.

10. There may be a relatively long interval between the recognition of the initial lesion and appearance of subsequent lesions.

Weinstein, Francis and Sprofkin report a case with lesions in the skull, pelvis and lungs; and yet they termed it eosinophilic granuloma, in contradiction to the terms laid down by the proponents of the disease, Lichtenstein and Jaffe.

John Raaf, in 1945, reported the case of a six-month-old child with lesions in the skull and fibula which responded to surgical extirpation.

Bailey and Freis report one case of so-called eosinophilic granuloma with lesions in the mandible, parietal bone and rib. Again, it is difficult to see how such multiple lesions can be classified "eosinophilic granuloma."

Salomon and Engelsher reported one case of a solitary supraorbital lesion which they called destructive granuloma.

In 1944, Osborne, Freis and Levin reported one case of so-called eosinophilic granuloma of bone in the temporal bone and mandible, in which the lesion of the temporal bone was severe enough to produce facial paralysis, deafness and labyrinthine symptoms. This case responded to X-ray therapy. It is difficult to see how this case differs substantially from the case reported by Rosenwasser as Hand-Schüller-Christian's involving the temporal bone.

Bakody, in 1946, reported a massive lesion in the skull diagnosed as eosinophilic granuloma, which was treated surgically and by postsurgical radiation and in which a tantalum prosthesis was inserted to take care of the skull defect.

M. H. Bass, in 1941, reported two children, one with a skull and one with a rib lesion; both cases responded to X-ray therapy. He reported the difficulty of pathologists in reaching unanimity of diagnosis, one calling the section eosinophilic granuloma and another Hand-Schüller-Christian's disease.

Dundon, Williams and Laipply, in 1946, reported five new cases of "eosinophilic granuloma," all in extremities.

Hamilton *et al.*, in 1946, reported nine cases of eosinophilic granuloma in various parts of the body.

Riley, in 1946, reported one case with rib involvement.

Solomon and Schwartz, in 1945, reported one case with a femur lesion.

Horwitz, in 1943, reported a case with a solitary tibia lesion.

Kernwein and Queen, in 1943, reported one case with a femur lesion in which they state that "eosinophilic granuloma is a condition incompatible with Schüller-Christian's disease clinically because of the single lesion and the universally favorable course."

Weidman, in 1947, reported a number of cases of "eosinophilic granuloma of the skin" and stressed that they are probably unrelated pathologically to eosinophilic granuloma of bone.

DEVELOPMENT OF THE CONCEPT OF ONE DISEASE.

Wallgren, in 1940, reported two cases in infants in which he showed quite conclusively that nonlipoid reticuloendotheliosis and Schüller-Christian's disease were identical and, further, that infectious reticuloendotheliosis, Letterer's-Siwe's disease and Hand-Schüller-Christian's disease are all different types of the same malady.

In 1941, Dr. Sidney Farber, of Boston, in a discussion on "Solitary, or Eosinophilic Granuloma," considered the entire subject as advanced by Lichtenstein and Jaffe and stated that in his study of 10 children he could find no difference between the pathological picture in eosinophilic granuloma and that of Schüller-Christian's disease or Letterer-Siwe's disease. Farber felt that none of these three conditions could really be considered an xanthomatous process or a manifestation of a primary alteration in lipid metabolism. He concluded that his studies do not lend support to the conclusion that eosinophilic, or solitary granuloma of bone is either a new or a separate disease entity. If this suggestion concerning the nature of these benign bone lesions is correct, caution must be exercised in the prognosis because of the possibility of later visceral involvement.

In 1942, Green and Farber reported 10 cases of benign bone destruction in which they were Roentgenologically and histologically indistinguishable from Hand - Schüller - Christian's disease. They, therefore, reaffirmed their previous statement that eosinophilic, or solitary, granuloma of bone was a variant of Hand-Schüller-Christian's disease and not a new entity.

In 1942, Gross and Jacox reported three cases and came to the conclusion that eosinophilic granuloma "is a reticuloendotheliosis and probably identical with those cases of Hand-Christian's disease which have been reported to have solitary lesions. It is also closely related to certain other reticuloendothelial hyperplasias."

In 1944, Versiani, Figueiro and Junqueira reported the case of a 50-year-old female with diabetes insipidus and a lesion in the femur. The histologic lesion in the femur showed a granuloma with two zones: *1.* an accumulation of eosinophiles giving the typical pictures of eosinophilic granuloma, and, *2.* lipophages, foam cells, arranged like a typical lipogranuloma as seen in Hand-Schüller-Christian's disease. They were, therefore, able to demonstrate in the same femur lesion the characteristic histologic findings of both eosinophilic granuloma and the Hand-Schüller-Christian type of xanthomatosis. They, accordingly, feel strongly that both are variants of the same disease.

In 1944, Engelbreth-Holm, Teilum and Christensen reported the detailed study of five children, aged two to nine years. All of these five children had been diagnosed at one time or another to have so-called eosinophilic granulomata of bone. The authors presented the following evidence to show that there was a gradual transition in the study of these cases from eosinophilic granuloma to Schüller-Christian's syndrome. In summarizing their work, they state:

"Clinically, transitions were formed from the solitary eosinophile granuloma through cases with several osseous foci to a generalized case with innumerable foci in the bone system together with diabetes insipidus, disturbance of growth and Roentgenographic changes in the lungs. In several cases where the disease commenced as a solitary granuloma, continued observation has revealed additional foci, so that in our material the number of foci increases with the length of the observation period.

"Morphologically, transitions were demonstrated from the lipoid-free eosinophile granuloma through granuloma with beginning lipoid phagocytosis with Touton cells to entirely xanthomatous tissue.

"After this, the concept eosinophile granulomas can no longer be maintained as a nosologic entity but must be considered a not altogether infrequent clinical monosymptomatic form of Schüller-Christian's disease that often heals without becoming generalized.

"Complete agreement is demonstrated between the various histological pictures in the cases of eosinophile granulomas here described and the various histogenic phases of Schüller-Christian's disease that may be characterized as follows:

"*1.* The hyperplastic-proliferative phase.

"*2.* The granuloma phase.

"*3.* The xanthoma phase.

"*4.* The fibrous (or healing) phase."

In 1946, Imler reported two cases from the therapeutic radiologic standpoint. He designated the disease itself "reticuloendotheliosis" and felt that the separation of Hand-Schüller-Christian's disease, eosinophilic granuloma and Letterer-Siwe's disease into specific disease entities was not valid on the basis of the evidence at hand. He felt that there were insufficient data to support the claim of a lipoid-metabolic disorder as the primary causative factor. He considered that these diseases were all variants of a hyperplastic reaction of the reticuloendothelial system; for that reason, he gave it the name of reticuloendotheliosis. In discussing diabetes insipidus, he felt that the stage of the disease process in the hypophysis and tuber cinereum would determine the response of the diabetes insipidus to deep X-ray therapy. If the infiltration is in the stage of fibrosis, no, or little, improvement could be expected.

In 1947, S. J. Thannhauser attacked the concept of eosinophilic granuloma of bone as advanced by Lichtenstein and Jaffe on the following grounds:

He showed first that eosinophilic aggregations had been described in this syndrome on many previous occasions. He called attention to the work previously quoted here by John Fraser, in which the earlier stage, so-called lipogranuloma, was manifested by endothelial proliferation and the accumulation of eosinophilic cells as well as giant cells. Thannhauser felt that the designation of the disease under discussion as eosinophilic granuloma was incomplete and therefore misleading, since "eosinophilic granuloma is not a disease entity but only a phase in the histologic aspect of a distinct clinical syndrome. The xanthoma cell formation in the later phases of the lesions is as characteristic of the disease as the reticulohistiocytic proliferation and the accumulation of eosinophiles in the earlier phases. If one does not prefer to use the older names, Schüller-Christian's syndrome or lipoid granulomatosis or normocholesteremic xanthomatosis, one should think of a classification which would at least refer to the principal two histologic phases of the granuloma; namely, a proliferative and eosinophilic phase and an xanthomatous phase. The designation of the localized and systemic disorder as eosinophilic xanthomatous granuloma seems to be more appropriate even if it does not embrace the reticulohistiocytic proliferation of the early lesion. Such a name would also prevent further misunderstanding in the conception and classification of this disorder.

"So-called eosinophilic granuloma of bone is certainly not a newly disovered disease entity but is the monosymptomatic form of the well known systemic granulomatous disorder in which histiocytes, eosinophiles and xanthoma cells are observed in the lesion at different phases."

Several months later, in December, 1947, Jaffe and Lichtenstein answered Thannhauser's criticism and defended the rationale employed by them in the setting up of the entity of eosinophilic granuloma. They pointed out the fact that "eosinophilic xanthomatous granuloma," which Thannhauser proposed as a comprehensive name for all cases, has the disadvantage that lesions of eosinophilic granuloma and of Letterer-Siwe's disease may be practically devoid of lipid; while, on the other hand, lesions of clinical Schüller-Christian's disease usually fail to show conspicuous eosinophilic reaction. "Whatever the anatomic interrelations may be, a case presenting an eosinophilic granulomatous lesion in one bone only (not a calvarial bone) without manifestations of general disease and in which simple curettage leads to complete healing of the condition has no clinical affiliation with cases of Schüller-Christian's disease. A simple analogy is in order. Miliary tuberculosis and active tuberculosis limited to a knee joint have the same bacterial cause, but they are decidedly different conditions from the clinical point of view. In the same way, though eosinophilic granuloma of bone and Letterer-Siwe's and Schüller-Christian's disease apparently represent different phases of the same basic disorder, there is a need for retaining at least clinical distinction between them."

They state further: "Even if it were to be demonstrated that some lesions of eosinophilic granuloma subsequently undergo transformation to lipoid granuloma, it still would make good sense on clinical grounds to segregate cases of eosinophilic granuloma, which is usually limited to one or a few bones and always runs a benign course, from cases of Schüller-Christian's disease, which often proves fatal and in which there are often pulmonary, cerebral or other visceral lesions in addition to skelétal involvement."

It is obvious, therefore, that, in 1947, Lichtenstein and Jaffe feel that the primary excuse for retention of the term "eosinophilic granuloma" is for a clinical distinction; and they, in fact, imply their agreement with the concept of a single disease which may have different clinical expressions during certain phases of the process.

Curtis and Cawley, in 1947, felt that the term "Hand-Schüller-Christian's disease" should be withdrawn from the diseases of lipid metabolism, the remaining members of which are Gaucher's and Niemann-Pick's. They feel that there are three related diseases; namely, Letterer-Siwe's, Hand-Schüller - Christian's and eosinophilic granuloma. They consider them all various forms of the same disease. They again mention the fact that Letterer-Siwe's disease is more common in infants; is severe; and is characterized by large skull defects, fever, hepatomegaly, splenomegaly, severe skin manifestations and high mortality. They go on to describe Hand-Schüller-Christian's disease as being characterized by skull lesions, diabetes insipidus, exophthalmos and skin manifestations in one-third of the patients. They conclude that eosinophilic granuloma is a disease of solitary bone lesions.

Strauss, in a recent article in 1948, gives three case reports and concludes: "It appears clear that eosinophilic granuloma of bone and its congeners, Hand-Schüller-Christian's disease and Letterer-Siwe's disease, represent merging clinical variations of an essentially similar pathologic process best described collectively by the term 'inflammatory histiocytosis'."

PATHOLOGY.

Hand-Schüller-Christian's disease was not clearly delineated from a pathological standpoint by either Hand, Schüller or Christian. It remained for Rowland, in 1928, to put the pathological understanding of this syndrome on a relatively definite basis. It was he who pointed out the similarity between the three observers; it was his original observation that this syndrome was an abnormality of the reticuloendothelial system in which the characteristic pathological finding was an abundance of so-called foam or xanthoma cells containing the specific lipid, cholesterol. It was upon this basis that he felt justified in distinguishing the disease from other lipoidoses such as Niemann-Pick's and Gaucher's. The triad of proptosis, diabetes insipidus and skull bony lesions was of interest and of some value from the clinical standpoint only; however, many pathologists insisted upon using the triad as a basis for a pathological diagnosis. Pathologically, this triad is not a necessary requirement for that stage of histiocytic granulomatosis in which xanthomatosis of the cholesterol type is the outstanding microscopic finding; nevertheless, on the basis of Rowland's work, which was a brilliant piece of investigation, xanthomatosis became a synonym for Hand-Schüller-Christian's disease; and the pathological picture, of necessity, included the triad, if possible, and the demonstration, microscopically of foam cells of the xanthoma type containing cholesterin.

The pathological picture of Letterer-Siwe's disease was never clearly defined; however, Abt and Denenholz described a *clinical syndrome* in which the outstanding features were high mortality, occurrence in early infancy, widespread reticuloendothelial proliferation throughout the body, fever, acuteness of the process, marked cutaneous manifestations, as well as visceral involvement by the reticuloendothelial dysplasia. Here the microscopic picture was described as being one where the outstanding feature was a so-called reticuloendothelial cell proliferation, the major finding being large clusters of reticulum cells with some giant cells and a very occasional foam cell plus scattered evidences of other cells, such as plasma cells, eosinophiles and polymorphonuclears. Here, again, we were given a picture which was primarily clinical and in which the pathological picture was not too clear-cut, as can be evidenced by the confusion existing in the minds of most capable pathologists, who, upon seeing a section of a lymph node or a bony lesion, will hedge for weeks and months before making a definite diagnosis. They will frequently state, "this appears to be a reticuloendothelial dyscrasia either of the Hand-Schüller-Christian or of the Letterer-Siwe type."

Finally, the proponents of the concept of eosinophilic granuloma state, as mentioned above, that they were describing a reticuloendothelial granuloma in which the predominant picture was histiocytic proliferation, but in which the characteristic accumulations of eosinophiles gave it a more or less individualistic status among these diseases, and on the basis of that finding, as well as the clinical impression that they were dealing with a monolocular disease primarily confined to the long bones, they advocated that the term eosinophilic granuloma be accorded true identification from the pathological standpoint.

In summary, therefore, we were told that Hand-Schüller-Christian's disease was a disease characterized primarily by xanthoma, or foam cells, containing cholesterin crystals; that Letterer-Siwe's disease was a disease characterized primarily by reticulum cell proliferation with occasional foam cells and occasional eosinophiles; and finally, the eosinophilic granuloma proponents, Lichtenstein and Jaffe, felt that they were dealing with a separate disease because, in addition to the histiocytic manifestation which was predominant, they also noted clusters of eosinophiles in their cases.

It is a widespread belief among pathologists today that one of the most difficult diseases to pigeonhole is the disease in which there is a question of xanthomatous or eosinophilic involvement of the reticuloendothelial system. It is probably due to no fault of the pathologists that this difficulty and confusion exists. It is quite obvious that the difficulty existed with the original descriptions of these three so-called clinicopathological entities; and the problem confronting the pathologist is how to make a diagnosis which has in itself never been firmly established in the original description of the disease.

THE UNIFYING CONCEPT OF HISTIOCYTIC GRANULOMATOSIS.

Numerous references to "reticuloendotheliosis" as a suggested term for the inclusion of all three of the above entities have appeared. They have been cited above. It is quite obvious, not only from the clinical but also from the pathological standpoint, that all three diseases are diseases of the so-called reticuloendothelial system. In the first disease classified according to acuteness, Letterer-Siwe's disease, the primary manifestation is reticulum cell proliferation. In the subacute form of the disease, Hand-Schüller-Christian's variety, the outstanding pathological finding is that of foam or xanthoma cells. And in the third or most benign and most chronic state of the disease, the eosinophilic granuloma, the outstanding pathological finding is eosinophilic aggregation around a predominantly histiocytic or reticuloendothelial response.

The essential pathological description in all three cases is that of granuloma formation. The granuloma in each case is of a nonspecific variety. Neither tuberculosis nor syphilis has ever been demonstrated as an etiologic agent in any of these cases. Virus infection has been suspected but never proved as an etiologic factor. The giant cells that appear in these cases are ordinary foreign-body giant cells and are not in themselves at all diagnostic of the type of granuloma. It seems logical to choose a term which will embody the basic pathologic alterations and to begin to eliminate the unnecessary confusion of three "diseases," the first two being named after their original authors, which, while indeed laudable and justifiable, is certainly not a boon to scientific terminology; furthermore, to the pathologist who is anxious to find a cytological and tissue method of description, a term embodying the basic cell form, namely, reticuloendotheliosis, is certainly to be desired.

The term reticuloendotheliosis has been advocated by Imler, Mallory and many others; however, a number of years ago it was pointed out quite logically and conclusively that the so-called reticuloendothelial system was a misnomer. The cell type involved was the histiocyte first described by Aschoff. The histiocyte by description was the cell participating in the various functions of the so-called reticuloendothelial system.

According to Maximow and Bloom, the term reticuloendothelium is not suitable, first, because it refers to only one of the representatives of the system, namely, the reticulum cell; second, because it creates the incorrect impression that the endothelium of the common blood vessels also belongs to this system. In speaking of the histiocytic system, Maximow speaks of a group of cells found in connective tissue and elsewhere, cells which are nonmotile but which are able to phagocytose particulate matter and to store foreign substances brought to them in colloidal solution. At first, phagocytosing and storing elements were found in different organs and tissues and were described under different names in the various places of the body. Maximow states, "In the loose, irregularly arranged connective tissue, they were called the resting wandering cells or clasmatocytes. In the lymphoid and myeloid tissues and in the red pulp of the spleen, the reticular cells were known to possess the same fundamental functional properties as the resting wandering cells; moreover, the flat cells which line the lymphatic sinuses of the lymph nodes and the venous sinuses of the bone marrow and the spleen were also found to act similarly. In the liver, the peculiar stellate or Kupffer's cells in the wall of the intralobular sinusoids and some of the cells lining the sinusoids of the suprarenal and the hypophysis were known to be endowed with a very marked power of phagocytosis and storage. The 'dust cells' of the lungs were called by this term because they showed the ability to take up particles of dust brought in with the inspired air. All these elements, although sometimes very dissimilar under physiologic conditions, react in a very similar if not identical way in response to certain stimuli. This led to the conclusion that they belong to one common large cell group which plays an important functional role in the body. The most appropriate name for these cells is histiocytes."

Accordingly, from the standpoint of correctness of terminology, it is proposed by the author that the term "histiocytic granuloma" be applied to the entire group of diseases variously characterized as Hand-Schüller-Christian's, Letterer-Siwe's and eosinophilic granuloma. The advantage of such a unifying concept is that it will clarify the apparently confusing picture illustrated not only in many of the bibliographic references but also in the cases cited above, where in a given patient it was possible to demonstrate clinical and histological features that would place the patient in either one, two or all three categories. The lack of true unity in each one of the three diseases and the lack of real differences between the three make it imperative that a more basic concept be employed, and that these three clinicopathologic divisions be maintained only as clinical names, if at all. Perhaps it would be wise to term the Letterer-Siwe's category the reticulum cell stage, or *acute histiocytic granuloma.* Hand-

Schüller-Christian's disease could then be termed the xanthomatous lipid-storing phase or *subacute histiocytic granuloma*. The solitary, chronic lesion of eosinophilic granuloma would then be termed *chronic histiocytic granuloma*. It would thus be possible to correctly classify many of these patients. Some of these children had not only diabetes insipidus, skull lesions and proptosis, to put them in the Hand-Schüller-Christian variety, but also had marked skin rashes and marked skeletal lesions to place them in the Letterer-Siwe's and eosinophilic granuloma classification. In almost every fatal instance it was possible to trace the pathological sequence of events relating to various stages in the histiocytic granulomatous response.

Pathologically, therefore, there should be only one disease, to the best of our present knowledge. Thus *histiocytic granuloma* could be termed acute, in the reticulum cell predominating phase, subacute, in the xanthomatous predominating phase, and chronic in the eosinophilic predominating phase. Clinical distinctions are of only little value. The disease has a markedly *random* and "helter-skelter" quality as evidenced by the geographic variety of the skull and extremity lesions. There are no two cases in which any true sequential or chronological order could be compared. The response of the histiocytic system is bizarre and unpredictable. Thus we see acute cases with and without bony or lymph node lesions. We see chronic cases with and without skeletal or sellar involvement. We see subacute cases with and without proptosis or skin lesions. Visceral lesions have been seen in every stage of the disease and have been absent in every stage of the disease, in varying patients.

The pathological findings in the various lesions are primarily due to pressure phenomena produced by the granulomatous process. It is interesting to note in the one available temporal bone section the resistance of the capsule of the bony labyrinth to invasion by the granuloma. All other bones in the body, including the tympanic ossicles, the annulus tympanicus and the entire mastoid and petron can be destroyed by this disease, but the bony capsule of the internal ear appears to be resistant.

DIAGNOSIS.

In making the diagnosis of histiocytic granulomatosis, for the sake of statistical value and in the interest of further research in the phases of this syndrome, it would be wise to attempt in each case, wherever possible, to allocate it to one of the three stages of the disease or to two of these, whenever that becomes necessary. The diagnosis of the classical reticulum cell, or the acute nonlipoid-storing phase, is in the case of the infant who develops a rapidly progressing disease characterized by multiple skeletal lesions, proptosis and visceral involvement, with early and quickly spreading cutaneous manifestations accompanied occasionally by diabetes insipidus, by profound anemia and by high mortality. The pathological picture by biopsy of the various tissues involved will usually reveal a widespread reticulum cell growth with occasional xanthoma cells in which there is no evidence of lipid storage. This stage will vary frequently and fuse almost *imperceptibly with the subacute xanthomatous stage of Hand*-Schüller-Christian type. Here the skin lesions will be less noticeable. There will be little, if any, visceral involvement, and the diabetes insipidus will be easily controllable in the vast majority of cases by the use of posterior pituitary extract and radiation of the sella. The skull lesions — all of which are identical in all three phases of the disease — will respond rather quickly to X-ray therapy. The response to X-ray therapy in the Letterer-Siwe type, or reticulum cell type, is usually poor.

The final chronic stage, that of "eosinophilic granuloma," is characterized most usually by monolocular lesions, but they may be skeletal as well as calvarial. The lesions will respond quickly to either surgery or X-ray; many patients have had uneventful recoveries by surgical curettage alone. Diabetes insipidus is usually not demonstrable, and skin lesions are only occasionally met with. The patient, while prone to recurrences in other parts of the skeletal system, usually makes an uneventful recovery following either surgical or deep X-ray therapy.

From the Roentgenographic diagnostic standpoint, there is practically no difference between the three phases, illustrating again the unity of the clinicopathologic picture. Thus Caffey states: "In the skeleton, the radiolucent granulomatous proliferations replace radiopaque bone and appear Roentgenographically as areas of diminished density in the flat bones of the skull, pelvis and shoulder girdle, and also in the tubular bones of the thorax and extremities. Involvement of the bones distal to the elbows and knees is practically unknown. These bony defects may be single or multiple; they are usually sharply demarcated and rounded or scalloped, and are of variable size.

"In cholesterol reticulosis (Schüller-Christian's disease and Letterer-Siwe's disease) the Roentgenographic findings are identical."

Under "eosinophilic granuloma," Caffey states: "Roentgenographically, the skeletal changes are identical with those of cholesterol reticulosis."

DIFFERENTIAL DIAGNOSIS.

A differential diagnosis on these patients must be made first from the standpoint of the skull lesions. The skull lesions are frequently confused with those of multiple myeloma and metastases from carcinoma. Lues and tuberculosis will occasionally produce this type of skull lesion. The outstanding feature in the behavior of this group is their usually quick response to deep X-ray therapy. Large tumor masses will disappear quickly and almost melt away within a question of days following administration of deep X-ray therapy to the calvarium, to the orbit and to the long bones as well.

The second differential diagnosis is the one that concerns the otologist specifically, and that is the differential diagnosis of temporal bone disease. It has been obvious to the writer in his examinations of these patients that the temporal bone lesion is frequently mistaken clinically for cholesteatoma. Indeed, several of these patients described above were originally diagnosed as having attic cholesteatomata. The fetid discharge, the frequent attic perforation, the evidence of bone destruction, conductive deafness and the Roentgen demonstration of trabecular destruction — all fit in very nicely with the diagnosis of cholesteatoma. What confuses the picture still further is that a smear taken for cholesterol crystals will almost always demonstrate them in histiocytic granuloma, particularly of the xanthomatous type, as well as in cholesteatoma. The differential diagnosis is best made by X-raying the skull for other lesions, by making a close search for diabetes insipidus by urine concentration studies, and by careful study of the eyes for evidence of early proptosis. The removal of a bit of granulation tissue for biopsy is an extremely valuable procedure and will frequently make the diagnosis. The response of the temporal bone to X-ray is just as dramatic as is the response to X-ray of the other cranial bones. The otorrhea will frequently cease, and the tympanic perforation will frequently heal entirely, following adequate deep X-ray therapy. The X-ray findings are of no value in distinguishing histiocytic granuloma from cholesteatoma unless a careful study is made of the other cranial bones by X-ray.

TREATMENT.

Inasmuch as the basic etiologic agent for the production of the histiocytic proliferation characteristic of the three phases of histiocytic granuloma is unknown, no specific therapy can be employed at the present time. The proponents of the virus theory have little or nothing in the way of proof for their thesis. The proponents of the hypercholesterolemic type due to increased cholesterol absorption and circulation or poor cholesterol excretion have little or nothing to say in their defense.

In short, we do not know anything at all about the etiology of this interesting type of granuloma; however, it has been observed that in most instances this lesion responds very quickly to deep Roentgen therapy.

The first acute phase of the disease, the reticulum cell type of Letterer-Siwe, usually does not show a good response to deep X-ray therapy. The response may be temporary or partial; but it rarely is of sufficient value to completely control the disease, and the patient usually succumbs to widespread granulomatous lesions which eventually interfere either with pulmonary, hepatic or cerebral function.

The subacute xanthomatous stage of Hand-Schüller-Christian shows a beautiful response to deep X-ray therapy in the vast majority of instances. There are, however, a large number that do not respond.

The third chronic low-grade eosinophilic phase very quickly responds to Roentgen therapy, but is as equally responsive to surgical extirpation. The judgment as to which to employ depends a great deal upon the site of involvement, mechanical factors that have to be considered and size of the lesions. An extremely large lesion might better be treated surgically with radiation reserved for the treatment of any residual swellings or recurrences.

Due to the limitations of space, it is impossible to report in detail on each of the 18 cases. The 18 cases are summarized in the chart at the end of the paper. The following three cases are somewhat typical and illustrate the main features of the disease.

CASE REPORTS.

Case 1. S. W.

History: This boy was first admitted to Children's Hospital on Feb. 16, 1944, at the age of two years, with a history of a chronic draining right ear of four months' duration, which had appeared spontaneously following a mild upper respiratory infection. The discharge was offensive and watery since onset. For the past month before admission, there had been a progressively increasing swelling in the right preauricular area.

Findings: Examination on admission revealed an unusually foul, profuse right otorrhea. The odor was foul but distinctly different from that noticed in cholesteatoma. The right external auditory canal was completely filled with a gray, friable, vascular mass. The tympanic membrane could not be seen. There was marked sagging of the superior canal wall. There was a large preauricular soft swelling which extended over the zygoma and obliterated the superior aspect of the postauricular crease. There were no other significant findings on physical examination. X-ray studies at that time showed equivocal findings in the right mastoid. Culture from the ear discharge revealed no growth of bacteria or fungi. Biopsy of a polypoid mass removed from the canal was reported as being too indefinite for a diagnosis.

Course: Because of the marked swelling and discharge, it was decided to perform an exploratory mastoidectomy on the right ear. Accordingly, on Feb. 19, 1944, a postauricular approach was made to the right mastoid; a large tumor mass was found to involve the entire temporal bone on the right side with complete necrosis of all mastoid cellular structure and complete exposure of the dura and the lateral sinus. The squama was almost entirely gone. A large defect extended posteriorly into the occipital bone and posteromedially toward the petron itself. Nowhere did the mass appear to encroach upon either the middle ear or the tympanic membrane. The superior and posterior bony canal walls were completely gone. The bulk of the tumor mass and necrotic bone areas was removed. The wound was closed with a T-flap, following the classical method of a radical mastoidectomy; however, no exenteration was performed in the tympanum inasmuch as it appeared to be uninvolved.

The material was distinctly different from the gross appearance of either a primary or secondary cholesteatoma; it resembled tumor tissue and had a very foul odor, differing markedly from the foul odor of cholesteatoma. Following surgery, a moderate otorrhea persisted. Histological sections were studied at length by the pathologist, who was unable to come to a definite diagnosis. His report read in part: "There appears to be some spaces which are lined with low keloidal type cells but they are indefinite, due to the presence of numbers of cells of large mononuclear or plasma cell type. In addition, occasional giant cells are scattered about. These for the most part are elongated, are relatively small, and have from six to 10 nuclei in them. The nuclei are somewhat elongated. Very few giant cells are noted which have round nuclei. On the margins of some of the necrotic areas some fibroblasts can be seen and there is a very definite inflammatory reaction in the form of occasional polymorphonuclears, some plasma cells and some large mononuclear cells; occasionally some eosinophiles are noted." The pathologist, upon further study, stated as follows: "This bears more of the aspect of tumor tissue than that of a chronic granulomatous reaction."

Because of the lack of adequate response to surgery and persistent otorrhea, deep X-ray therapy was started empirically with no definite diagnosis. Patient was discharged from the hospital and followed on the outside by a pediatrician, as well as by the author.

In July, 1944, approximately five months following his discharge from the hospital, his mother noted that he suddenly began to crave large quantities of fluids. This continued until his next admission to the hospital in October, 1944. He drank huge quantities of water and urinated 20 to 25 times daily. In August, 1944, his appetite became poor and he began to lose weight. On readmission, the only positive findings were a healed right mastoid wound with a normal tympanic membrane and a large, widened and deformed external auditory canal. The specific gravity of the urine was 1.003; the blood plasma cholesterol value was 229 mg.

per cent. Further X-rays of the skull revealed an extensive defect consistent with xanthomatosis, involving the floor of the middle fossa. It was the impression that he had very definite diabetes insipidus, and further X-ray therapy to the pituitary region was advised. He was then discharged from the hospital and followed as an outpatient. He has been seen regularly by the writer since that date and was recently examined in September, 1948. During the past four years he has maintained excellent nutrition. His diabetes insipidus was controlled after about four months of Roentgen therapy to the sella turcica region. During these four years, however, he has had at least six episodes of painless swelling in the right parietal, temporal and occipital areas. He has had several areas of regrowth of redundant granulomatous tissue into the external auditory canal. Each episode of swelling of the skull was immediately followed by one or two suberythema doses of deep Roentgen therapy. In every episode so far during the last four years each mass has disappeared.

A biopsy of a piece of tissue removed in July, 1947, was described histologically as follows: "The specimen is made up of a loose network of fine connective tissue; in this are infiltrated numerous inflammatory cells of all types. In some areas, polymorphonuclears predominate; in other areas, numerous eosinophiles are present. There are also lymphocytes, plasma cells, eosinophilic plasma cells. In one margin of tissue there are some quite large cells which have foamy cytoplasm in abundance. The cytoplasm appears to be vacuolated. Occasional mononuclear cells are

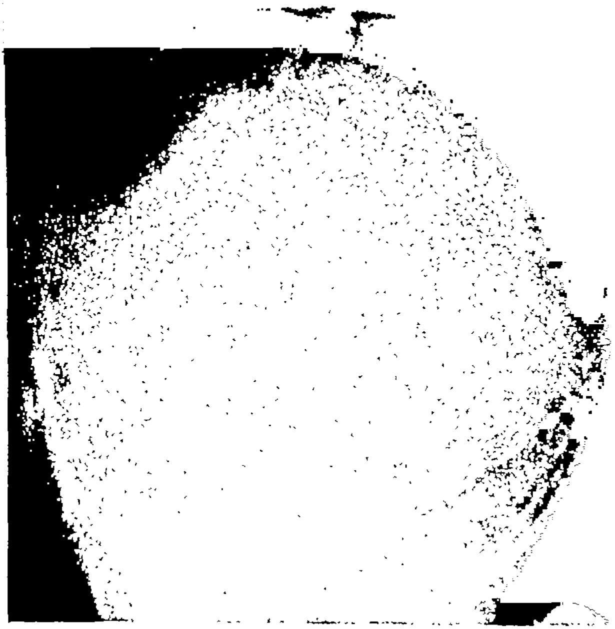

Fig. 1. Case 1. S. W. Posteroanterior view of skull showing extensive lesion in the right temporal bone with replacement of all of the pneumatized area by granuloma.

present. The foam cells are consistent with cells seen in xanthoma. These are large mononuclear phagocytic cells which have picked up fat."

Discussion: This patient has now been followed for almost five years. He presented two findings, chronic otorrhea due to mastoiditis and diabetes insipidus. He had no proptosis and no other significant findings. A great deal of difficulty was encountered in making a pathological diagnosis because of the great variation in histological structure in the specimens examined; however, in retrospect, it is easy to see how one could have made a diagnosis either of the subacute stage of histiocytic granuloma (Hand-Schüller-Christian's disease) or eosinophilic granuloma, the chronic stage. As noted above, sections revealed not only typical reticuloendothelial formations but large clumps of eosinophiles and large groups of xanthoma, or foam cells; however, in view of the clinical findings of diabetes insipidus and a huge temporal bone lesion followed later by local multiple recurrences, it is obvious that this case does not fit into the chronic, or eosinophilic granuloma, pattern but rather into the subacute, or Hand-Schüller-Christian, pattern of histiocytic granuloma.

Case 2. W. E.

History: This patient was first admitted to Children's Hospital on Sept. 16, 1936, at the age of two years and four months. In March, 1936, when he was less than two years old, he received an injury to the left orbit, following which the eye became prominent and red.

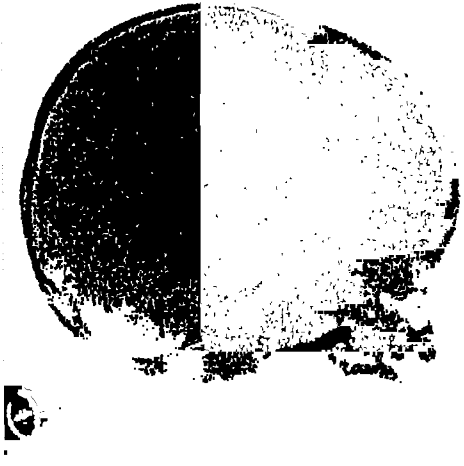

Fig. 2. Case 2. W. E. Patient on admission shows a very slight beginning skull lesion. [illegible]

Findings: Examination at that time revealed a marked proptosis of the left eye with a widened palpebral fissure. There were no other abnormal findings. There was a slight swelling of the lateral margin of the orbit, and the bony lateral orbital wall could not be palpated. X-ray at that time revealed a destruction of the outer rim of the left orbit, apparently from pressure. The orbit was enlarged.

Course: An exploration of the left orbit was performed. Some of the abnormal tissue in the orbit was sent for biopsy. The report by the pathologist contained the following statements: "There are many polyhedral and round tumor cells within the vesicular nuclei and pink staining cytoplasm. There are also many large cells which contain pigment giving a positive test for iron. There are also a number of giant cells containing from two to six or eight nuclei. A few of these, at least, are typical foreign body giant cells." There were many other remarks indicating real difficulty in establishing a real pathological diagnosis and the final statement was, "Tumor tissue, moderately malignant, type of primary site unknown."

The patient was then discharged from the hospital and X-ray therapy was advised. He was definitely improved on X-ray therapy and continued in good general health. He was, however, readmitted to the hospital on Aug. 9, 1937, for further study because of a beginning fullness in the right temporal fossa. In view of the appearance of a new skull lesion, further X-rays were done, and these showed beginning erosion in the outer right orbit plus several eroded areas in other parts of the skull. At this time, the Roentgenologist suspected that we were dealing with xanthomatosis.

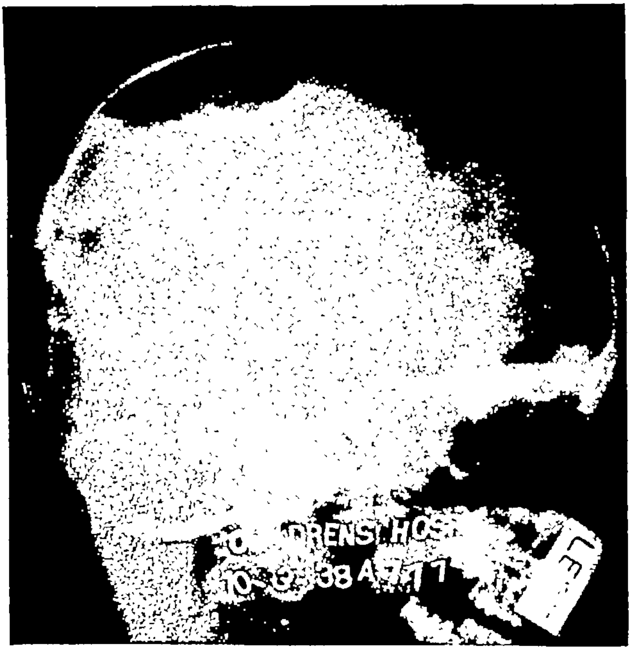

Fig. 3. Case 2. W. E. Marked extension of skull lesions.

During this hospitalization for study, a request was made for otolaryngologic consultation, since neither tympanic membrane could be visualized because of debris and cerumen. The writer saw the patient on Aug. 21, 1937, for the first time. Examination at that time revealed the following findings: The left ear canal contained a peculiar type of granulation tissue which obstructed a view of the left tympanic membrane. The right tympanic membrane was visualized and was perfectly normal in every way. Within two days of observation, the granulation tissue noted in the left ear increased greatly and appeared to be actually polypoid. These polypoid granulations were then removed and at time of surgery were noted to be of intratympanic origin. Foul purulent discharge was noted following removal of the polypoid granulation tissue. The report of the pathologist on the biopsy of these polypoid areas was that of definite xanthomatosis. "The specimen consists of bits of tissue, the typical cells of which are large, pale, foamy cells having vesicu'ar nuclei. Many of these cells are multinucleated and show vacuoles in their cytoplasm. Numerous acute inflammatory cells including polymorphonuclears, lymphocytes and plasma cells are found infiltrating the area occupied by the large cells and forming a marginal false membrane about the greater part of the section. The remaining portion of the periphery is covered by a small piece of degenerated squamous epithelium. Diagnosis—Xanthomatosis."

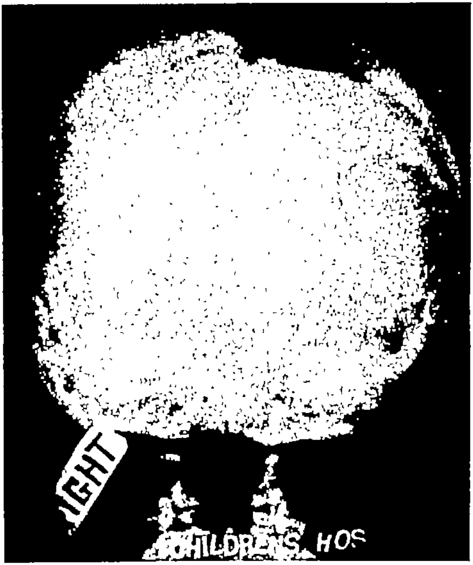

Fig. 4. Case 2. W. E. Further extension of skull lesions.

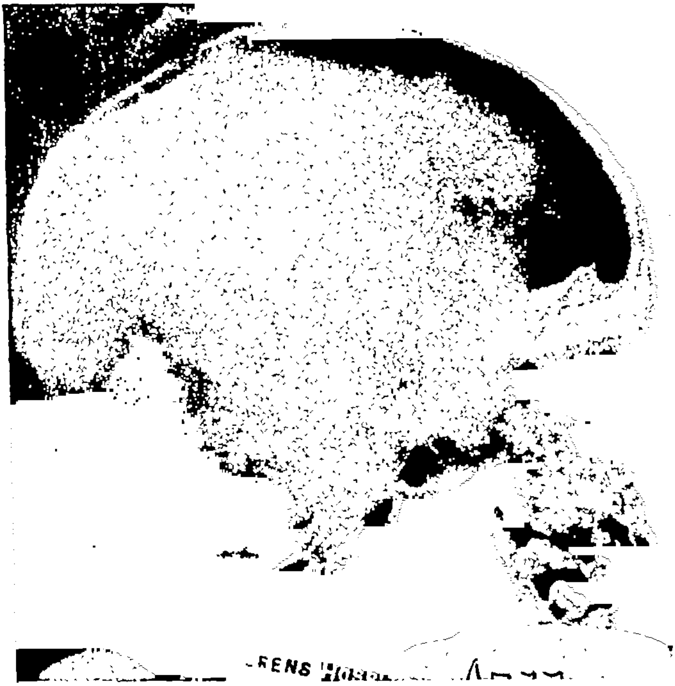

Fig. 7. Case 2. W. E. Skull lesions have almost completely filled in. X-ray taken one month before death.

complete absence of bony structure at its center. In a round area measuring about 1.3 cm. in diameter, very thin bone then appears and this gradually increases in thickness until it reaches the margin of the area. In the flat spot in the temporo-occipital region there is bone covering the entire area, but it is thinned down to 1 mm. in thickness. There is no evidence of yellow discoloration at any of these sites. When the calvarium is removed and the dura is incised, the brain is exposed and it shows marked subarachnoid edema. Brain is rather tense. Coronal sections of brain show no evidence of dilatation of ventricles. *On sections through the medulla, however, there are irregular circumscribed yellowish areas.*

"About the sella there is distinct yellow discoloration to the bone; and when the sella is chiseled away to remove the pituitary, there are yellow foci both in the bone and in the dura about the pituitary. When the middle and inner ears are opened, no exudate is noted; but there are distinct brilliant yellow foci in the bone on both sides. This is more marked on the left.

"The pituitary itself is not remarkable. About it, however, and the underlying skull and in the dura about it, there are numerous large, pale, foamy-appearing cells which are so numerous in the connective tissue that it seems to be much thicker than normal dura, and the cells are also infiltrated into the periosteum. The bone itself shows no distinctive

1.		Name	1. R.E. M	2. S.W. M	3. W.E. M
2.		Hospital	CH	CH	CH
3.		Hospital No.	29-3057	44-3889	36-2929
4.		Age of onset	3 years	20 months	23 months
5.		Duration of observation	19 years	4½ years	6¼ years
6.		Outcome—alive or dead. Age.	Alive and well—age 22	Alive and well—age 6	Died—age 8
7.	SKULL LESIONS	Frontal	X	O	X
8.		Temporal with otorrhea	O	X	X
9.		Temporal without otorrhea	X		
10.		Parietal	X	X	X
11.		Occipital	X	O	X
12.		Orbit	X	O	X
13.		Sella	O	X	X
14.		Proptosis	X	O	X
15.		Diabetes insipidus	X	X	X
16.		Skeletal lesions	X pelvis	O	O
17.		Skin lesions	O	O	O
18.		Visceral lesions	O	O	O
19.		Abnormal lab. findings	O	Cholesterol—227 mgm. % Urine sp. gr. 1.003	Urine sp. gr. 1
20.		Miscellaneous findings	O	O	Central nervo system diseas
21.		Surgery	O	Modified radical mastoidectomy	Orbital decom polypectomy a
22.		Radiation	X	X	X
23.		Histological findings	O	Eosinophilia, xanthoma and chronic granuloma	Xanthomatous
24.		Pathologist's diagnosis	O	Xanthomatosis	Xanthomatosi
25.		Comment	1st clinical diagnosis: Multiple myeloma	1st pathological diagnosis: "tumor" 2nd: Xanthomatosis	1st pathologic "malignant tu 2nd: Xanthom

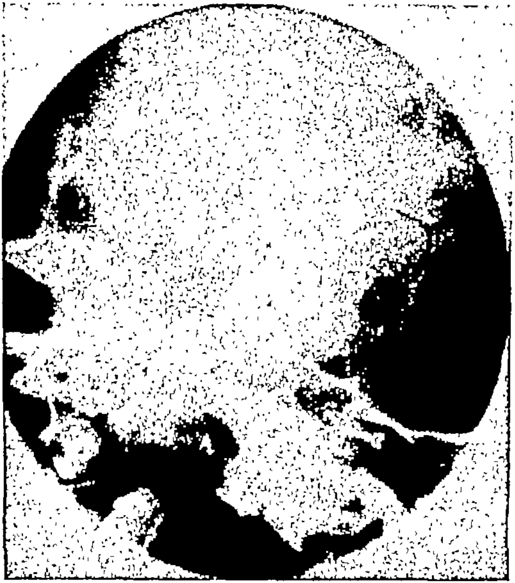

Fig. 8. Case 2. W. E. Skull showing erosion of anterior clinoid process of sella turcica and showing lesion in temporal bone.

abnormality; the bone marrow, although scanty, is negative. Other sections of skull show evidence of bone atrophy, and in many of the bony spaces there are numerous large foam cells similar to those described above. These areas of foam cell infiltration correspond to the areas of yellow discoloration described in the gross.

Discussion: This patient was followed for six years. He gave the clinical findings of bilateral proptosis, bilateral temporal bone lesions, diabetes insipidus, widespread generalized skull lesions, and terminal cerebral involvement. In view of his more or less chronic course, he must be considered to be a subacute fatal form of histiocytic granuloma in which death resulted from cerebral involvement even though all of the skull lesions and diabetes insipidus were completely cleared following deep X-ray therapy. The final impression, therefore, is that we were dealing here with subacute histiocytic granuloma of the xanthomatous type falling most readily into the Hand-Schüller-Christian designation.

Case 3. S. S.

History: This three-year-old white female was admitted to Children's Hospital on Aug. 30, 1946, with the following history: At the age of

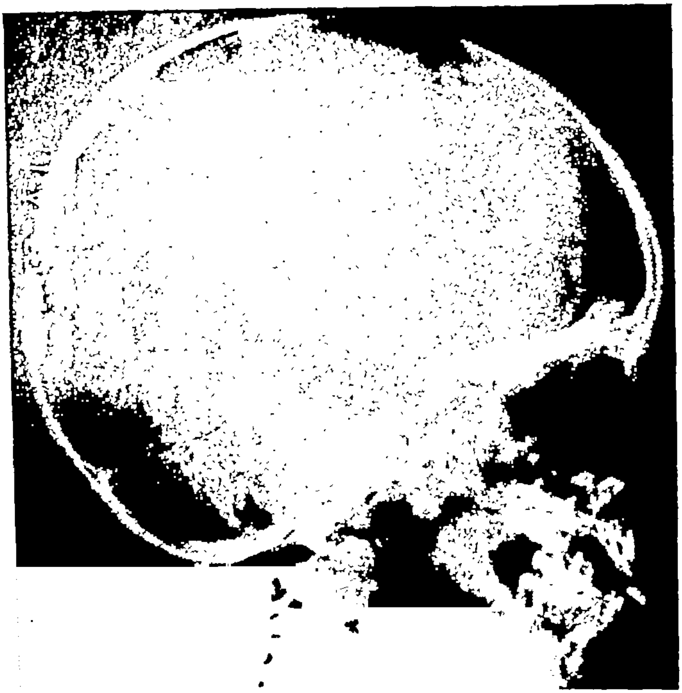

Fig. 9. Case 2. W. E. Lateral view of skull showing progression of parietal and occipital lesions.

11 months, in July, 1944, the child developed spontaneous bilateral draining ears which did not follow either upper respiratory infection or contagious disease. At the same time, the skin over her chest and under her right arm became raw and started to drain serous fluid. This clinical picture persisted until November, 1944, when she developed a lump over the right forehead, which increased in size over a period of several weeks. It was incised by a local doctor, but did not clear up. At about this time, the skin lesions which had started several months before had become larger, so that they now spread to involve the entire body and scalp. The lesions were crusted and were occasionally weeping. At this time a diagnosis of probable xanthomatosis was made, and she received X-ray therapy for the tumor of the skull, the mastoiditis, and the skin lesions. Because of the continuation of the otorrhea, in November, 1945, a bilateral simple mastoidectomy was performed elsewhere. The ears, however, continued to drain in spite of the fact that the mastoid wounds healed nicely. X-ray therapy was continued to the skin and the scalp lesions. Following surgery to the mastoid regions, the child began to drink constantly and urinated profusely. She was then treated with posterior pituitary powder by insufflation, with some improvement; however, from that time until July, 1946, her course was progressively downhill. The skin lesions became more advanced, the tumors of the scalp became greater, and the diabetes insipidus persisted. In July, 1946, jaundice appeared, and the child was then noted to have diarrhea with-

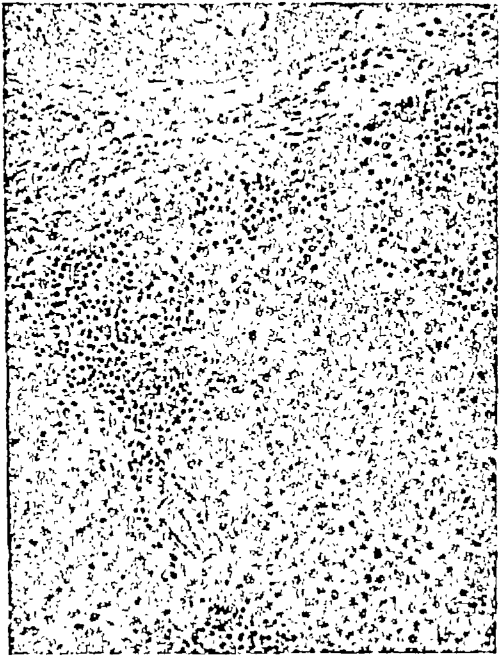

Fig. 10. Case 2. W. E. Section of base of skull in hypophyseal area showing large accumulation of pale xanthoma, or foam, cells characteristic of subacute histiocytic granuloma (Hand-Schüller-Christian's disease).

out blood. Her urine became dark about this time, and the eyes became rather prominent. A protrusion of the abdomen was also noted, and her general condition became poor.

Findings: On admission the child appeared to be in great distress. The temperature was 99.8°, pulse 120, respirations 28. The body weight was 24¾ pounds (the normal for that age and height should have been 31 pounds). The height was 35½ inches (the normal for her age was 36½ inches). She appeared to be an emaciated, chronically ill, white female of three years. Examination of the head revealed a defect in the right frontal area, which was soft to palpation. It measured 2 x 2 cm. A similar defect was noted over the left parietal area, which measured 2 x 3 cm. and was tender. There was a slight left exophthalmos; both sclerae were markedly icteric. Both ear canals were filled with a foul seromucoid discharge; a marked crusting skin lesion obscured both

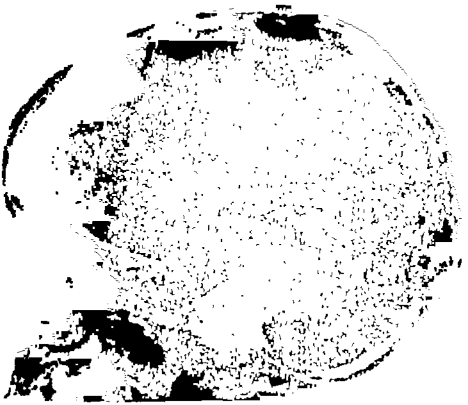

Fig. 11. Case 3. S. S. Lateral view of the skull showing marked geographic erosions in the entire skull.

quent eosinophiles are noted in the sinusoids, also some larger mononuclear cells with homogenous pink cytoplasm."

Skull lesions: "On one side there is a wide band of typical foam cells with a few capillaries and little other tissue, then a band of collagenous tissue, next a wide layer of young fibrous tissue and a heavy infiltration of medium-size mononuclear cells with pink homogenous cytoplasm. There is then a band of necrotic cells and another fibrous and mononuclear cell layer. In this last layer there are numerous foam cells, also large mononuclear cells with pink granular cytoplasm. Some of these become quite large and form multinucleated giant cells. In addition, there is a light infiltration with polymorphonuclears and lymphocytes."

Pituitary: "The anterior lobe is identifiable, consisting of acinus-like structures composed of bright red-staining cells, chromophiles, and many purple-staining cells. This ends more or less abruptly in a curving line in a mass of closely packed cells with spindle-shaped nuclei and an abundant narrow, fusiform, irregularly directed cytoplasm. There is one small area of recent hemorrhage into this tissue. A few lymphocytes and a rare polymorphonuclear and eosinophile are noted in this tissue. In some areas there are accumulations of large mononuclear cells with homogenous pink cytoplasm. These are similar to those seen elsewhere A rare foam cell is also present."

Fat analysis by Dr. H. J. Deuel, Jr., *Professor of Biochemistry*, U. S. C. School of Medicine: "The data show an undoubted increase in the lipid content of the liver and, of course, the characteristic high cholesterol content of the skull. The lipid content of the lungs is also tremendously high along with the cholesterol."

tous and contained, in various fields, representations of every phase of histiocytic granuloma. There were numerous areas of marked reticulum cell formation; there were areas of widespread foam cell, or xanthoma cell, formation; and finally there were many areas with large clumps of eosinophiles such as are frequently found in eosinophilic granuloma.

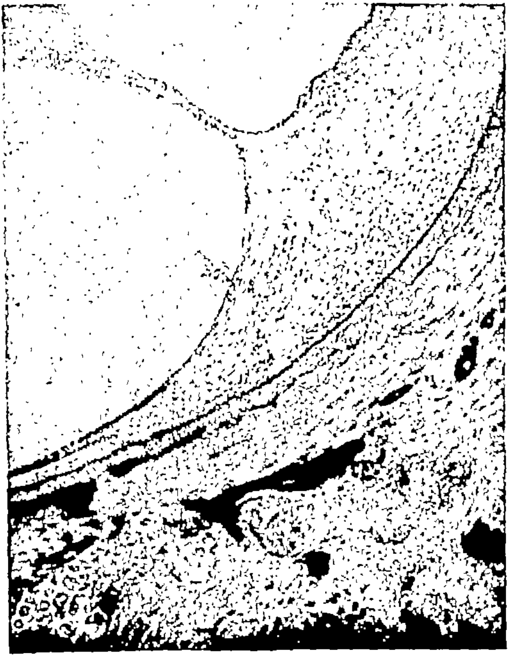

Fig. 13. Case 3. S. S. Temporal bone section showing reticulum cell involvement of pneumatized areas but no involvement anywhere in the scalae. The bony capsule of the labyrinth was apparently able to prevent infiltration of the internal ear by this subacute histiocytic granuloma.

Accordingly, in this one temporal bone it was possible to demonstrate every histological phase of the disease, acute, subacute and chronic, indicating the fact that this was undoubtedly a very severe process which had the opportunity to develop every histologic characteristic of the disease. Interestingly enough, in no section could invasion of the bony capsule of the labyrinth be demonstrated. The cochlea throughout its

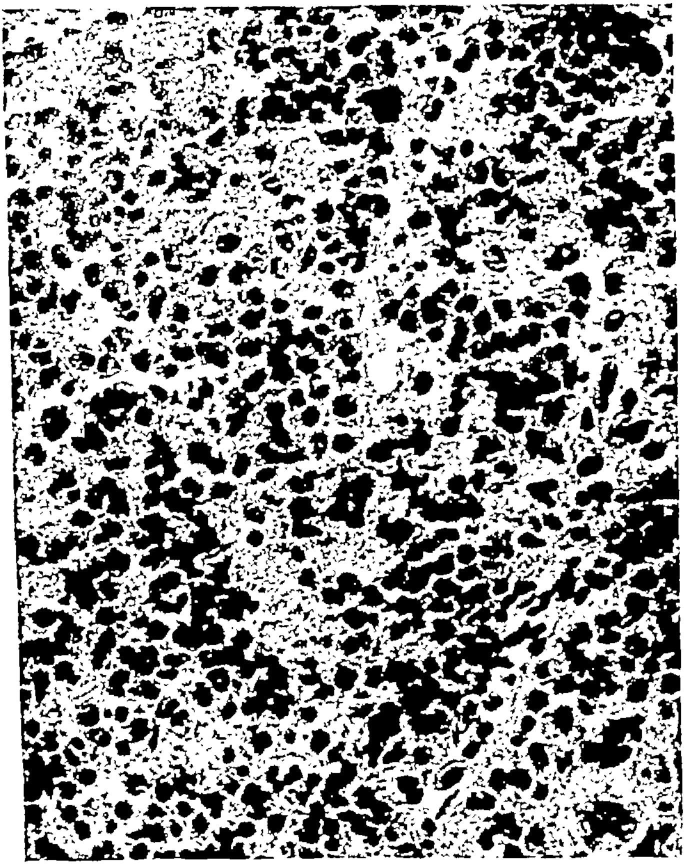

Fig. 14. Case 3. S. S. Section showing numerous eosinophiles, plasma-like cells, histiocytes, and occasional xanthoma-type cells.

coils was essentially normal, as were the various elements of the vestibular apparatus. The infiltration was entirely within the cancellous bone structure and cellular trabeculae of the temporal bones.

Discussion: This case is of great interest in that, first, it showed a fulminating acute process which ended fatally in approximately two years after onset. The disease was characterized by almost every manifestation of histiocytic granuloma, starting with the skin lesion and going on to rapid progression, including skull lesions, proptosis, diabetes insipidus, bilateral temporal bone lesions, hepatomegaly, splenomegaly, icterus, and finally pulmonary involvement. It was possible in this patient to demonstrate all of the histological characteristics of the disease in the acute, subacute and chronic phases. It is also noteworthy

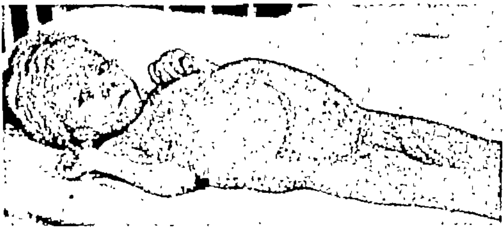

Fig. 15. Case 3. A. S. Photograph showing lateral view of body several days before death. Note the characteristic purpuric seborrheic skin rash over the forehead, scalp, palms, chest and abdomen. Note also the enlarged abdomen, enlarged liver and enlarged spleen as indicated by markings on abdomen.

that response to X-ray therapy and surgery was poor and that, in spite of all treatment, the patient maintained a steady downhill course and finally died.

CONCLUSIONS.

1. Three diseases heretofore considered specific entities, namely, Letterer-Siwe's disease, Hand-Schüller-Christian's disease and eosinophilic granuloma, have been shown by many investigators to be probably different phases of the same basic disease process.

2. The basic disease process is a dysplasia and hyperplasia of the histiocytic or so-called reticuloendothelial system, which creates granulomata which by predilection occur in certain parts of the body. The term "histiocytic granuloma" is proposed as a basic entity to include the three phases.

3. The acute stage of the disease, the reticulum cell type of Letterer-Siwe, is characterized by its occurrence in early infancy; the rapid involvement, not only of the calvarium but of the other skeletal areas; rapid visceral involvement; and early cutaneous manifestations. The mortality is extremely high and the response to treatment by radiation therapy is extremely poor.

4. The subacute, or xanthomatous, stage of Hand-Schüller-Christian's is best typified by the clinical triad of proptosis, skull lesions and diabetes insipidus due to involvement of the sella turcica. In this stage the xanthoma, or foam cell, is the outstanding pathological characteristic. The prognosis is relatively good, the patient's response to radiation being on the whole quite excellent.

5. The chronic form of the disease characterized by eosinophilia as well as histiocytosis and previously termed eosinophilic granuloma responds equally well to surgery or radiation. It is most usually confined to the long bones but may occur in the calvarium.

6. Eighteen cases are reported for the first time, in many instances showing impossible attempts to categorize the cases into any one of the three phases previously called separate diseases. In most instances, manifestations of two and occasionally of three phases can be found in the same patient.

7. Of statistical interest are the following facts:

a. Of the 18 cases reported, 11 were males and seven females.

b. No "racial" or geographic factors were found to predispose to the disease.

c. Of the 18 cases reported, 11 had temporal bone lesions, 10 with chronic otorrhea.

8. It is important for the otolaryngologist to be highly conscious of the relative frequency of this disease inasmuch as it may very frequently simulate malignant tumors, lues, tuberculosis and other diseases. The patient with an unexplained proptosis, an unexplained diabetes insipidus and a temporal bone lesion of the chronic otorrhea type should be investigated by appropriate Roentgen studies for the possibilities of a histiocytic granuloma.

The response to treatment in this disease, if recognized early, is usually good. Consequently, a plea for early diagnosis must complete these conclusions.

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NASOPHARYNGEAL IRRADIATION AND HEARING ACUITY: A FOLLOW-UP STUDY OF CHILDREN.[*][†][‡]

STACY R. GUILD, Ph.D.,
Baltimore, Md.

My part in this Symposium is to report observations made in a follow-up study of school children. Many persons have participated, at one time or another and in various ways, in the collection of the observations on which this report is based, and I wish at this time specifically to acknowledge this fact and to express my appreciation to all the members, past and present, of the staff of the Department of Otolaryngology who have, by their carefully made hearing tests, good physical examinations, irradiation treatments, etc., made this study possible. Throughout the study I have participated actively in the collection and in the analysis of the observations. This report is based on analyses made mostly by me, and I assume sole responsibility for the content of the report.

The persons whose records form the basis for the present report were first examined in the Otological Research Laboratory of the Johns Hopkins University School of Medicine during the school year 1939-1940, in connection with the study of the hearing of 1365 white school children that was reported at the 1940 meeting of the American Otological Society.[12] During the next two years a large number of the children were re-examined periodically; a report of this two-year follow-up study was made at the 1942 meeting of the Ameri-

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[*] Presented, by invitation, in a Symposium on Irradiation of Lymphoid Tissue in the Nasopharynx, at the Fifty-fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11, 1949, Chicago, Ill.

[†] From the Otological Research Laboratory, the Johns Hopkins University School of Medicine.

[‡] This study was aided by grants to Dr. S. J. Crowe from the John and Mary R. Markle Foundation.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 21, 1949.

can Otological Society.[9] The shortage of departmental staff incident to the entry of our country into World War II interrupted the program of periodic re-examinations that had been planned. After the war, during 1946 and the first half of 1947, re-examinations were made of all the children, irrespective of whether or not they had been treated, who could then be located and persuaded to return to the laboratory for re-examination. The present report is based on comparisons of the records of the first and of the last examinations made of the children who actually returned in 1946 or 1947.

The shortest interval between the first and the last examinations of the children whose records are used in this report was 6 years, the average interval was slightly over 6½ years. The average age of the children at the time of their first examinations was 9½ years, and at the time of their last examinations slightly over 16 years. The age range at the time of the first examinations was from 8 to 13 years; at the last examination it was from 14 to 21 years.

Selection of the children for examination originally, in 1939-1940, was not on the basis of hearing or of present condition or past history of the nasopharynx or the ears, but was entirely on the basis of enrollment in certain classes of certain schools. The parents of all the children who were then attending regular classes in the third, fourth, fifth or sixth grades of one or another of six Baltimore schools were asked to permit their children to participate in the study. The only departure from a strictly random selection of white children of that age group is therefore the one imposed by the requirement that they be attending regular classes; this automatically excluded certain categories of seriously handicapped children, such as the mentally defective, the blind, the severely crippled, and children with extreme degrees of impaired hearing. The effect on the present report of the exclusion of the latter group will be discussed later.

All examinations were made at the hospital, under suitable conditions for careful work; none was made at a school. All the examiners were well qualified for the parts of the examinations they made. Most of the irradiation treatments were given by Dr. S. J. Crowe personally. The hearing tests, the reliability and accuracy of which particularly concern the credibility of this report, were all made in one or the other of the two "sound-proof" rooms at the laboratory. At each examination, a Western Electric Co. 1-A audiometer was used to determine the thresholds, by air conduction, for tones of 14 frequencies over the range from 32 to 16384 cycles per second; the charts show the frequencies routinely used in the tests. The hearing acuity for spoken voice was also tested at each examination, by means of a W. E. Co. 4-A phonographic audiometer and the two-digit records; and at each examination Weber, Schwabach and Rinne tests were made with a 512 d.v. steel tuning fork. The opposite ear was masked, routinely, during the making of the phonographic, the Schwabach and the Rinne tests; the masking noise was generated in a headphone by a calibrated masking device, locally built, that has been used in the laboratory for many years. Suitable masking was also used, whenever indicated, during the testing with the pure-tone audiometer. The audiometers (two of each kind), the tuning forks and the masking devices were meticulously maintained in proper condition for use throughout the period of time covered by this study. In brief, the last examinations of the children in this study were made under conditions strictly comparable to those that prevailed at the time of the first examinations. This point is stressed because otherwise some of the changes in hearing acuity that occurred might be regarded as apparent rather than as real ones.

CHILDREN WITH GOOD HEARING; NOT TREATED.

The majority of the children examined had good hearing, as would be expected from consideration of the method of selection used. The inclusion of this group in the follow-up study has proved to be important, for three reasons: (1) it has made possible the study of normal ageing changes in hearing acuity during the childhood years; (2) it has provided information with respect to the effect of nasopharyngeal lymphoid tissue on the normal ageing changes that occur in hearing acuity during childhood; and (3) it has provided a baseline that can be used in the evaluation of the significance of changes that occurred in the hearing acuity of the children who had impairments limited to high tones and who were treated by nasopharyngeal irradiation. It is for the latter reason that the data about this group of untreated children are included in this Symposium on the effects of nasopharyngeal irradiation.

Of the children who returned in 1946 or 1947 for re-examination, a total of 259 met the requirements set up for inclusion in the group in which normal ageing of hearing acuity might be studied; namely, that at the first examination both ears had good hearing or at most a slight impairment for some tones of the very high frequencies, and that during the time between the first and the last examination no treatment of the throat, either irradiative or surgical, was received, either from members of our own organization or from physicians elsewhere. None of the group had had any nasopharyngeal irradiation previous to the first examination, but about half of the children had been submitted to throat surgery, either a tonsillectomy or a tonsillectomy and adenoidectomy, before they entered the study.

The changes in hearing thresholds that occurred in the entire group of 259 children (518 ears) during the period of observation are summarized in Fig. 1. In this, as in all but

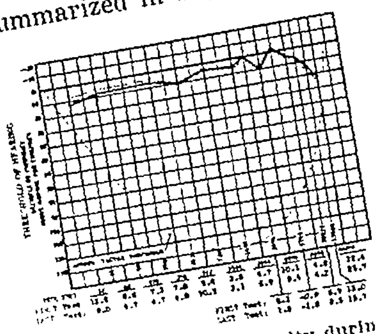

Fig. 1. Normal age changes in hearing acuity during childhood, based on tests of 259 untreated children (518 ears) who were re-examined after six years or longer. As in all except the last figure in this paper, the solid line shows the average thresholds of the first audiograms and the dotted line the average thresholds of the last audiogram, and the numerical averages, to the nearest tenth of a decibel, are stated below the chart.

one of the charts used in the report, the audiogram shown by the *solid* line represents the arithmetical averages, or the means, of the air-conduction thresholds for the respective tones at the time of the first examination, and the audiogram shown as a *dotted* line represents the corresponding averages for the last examinations. The actual averages, to the nearest tenth of a decibel, are given in tabular form below each audiogram chart, for the benefit of persons who may care to study the data more exactly than can be done by inspection of the graphic audiograms. It is impossible to show slight differences in thresholds on a standard audiogram form except by using lines so thin that they are not reproduced clearly on the printed page.

Before proceeding to inspect the figures, a few words are in order about the use of averages to present the data of this study. I am well aware of the limitations of the method and of the pitfalls into which averages may lead one when the raw data cover a wide range of variation. For instance, it means nothing to have the average of the ages of the persons in a home for the aged and in a kindergarten class be the same as the average of the ages of the firemen in the city. But when the range of variation for similar items in the raw data is small, the method of averages is satisfactory for most purposes. I believe that to be the case in the present instance; the age range is small, the differences in length of the intervals between the first and last examinations were small compared to the total interval, and the children within each group or subgroup that has been made of the material had only small differences in hearing acuity, compared to the possible range of difference. Certainly this simple method reveals the essential story so well that it has not seemed worth while to use other statistical techniques.

Inspection of the average audiograms displayed in Fig. 1 shows that, for the group of 259 untreated children (518 ears), hearing acuity improved slightly for low tones during the period of observation, became slightly worse for very high tones, and remained almost unchanged for tones of the frequency range from 512 to 8192 cycles per second. The improvements for the two lowest tones tested (32 and 64 cycles) were somewhat more than for the lowest tones generally used in audiometric testing, namely, those with the frequencies of 128 and 256 cycles. The reasons for believing that the shifts in thresholds recorded for this group of children represent the normal ageing changes are stated elsewhere in this report.

Only one of the breakdowns that have been, or can be, made of the observations recorded for the group of 259 untreated children, will be presented today. This is the breakdown made on the basis of the recorded appearances of the tubal orifice regions of the nasopharynx, as viewed through a nasopharyngoscope, at the times of the first and of the last examinations, respectively. To avoid the making of subgroups too small to be significant, and at the same time to help avoid confusion in the interpretation of descriptions by different examiners, the classification for each examination has been restricted to two categories: both tubal orifices normal, and one or both tubal orifices abnormal.

The average audiograms for the four subgroups thus made of the 259 untreated children are shown in Fig. 2, parts A to D.

The largest subgroup is the one in which the children had abnormal tubal orifices at the time of the first examination and normal tubal orifices at the time of the last examination. The children, in other words, had had a spontaneous regression of nasopharyngeal lymphoid tissue during the transpubertal period covered by the study. This subgroup consists of 130 children, or almost exactly half of the total group of untreated children. The average audiograms of these 130 children (260 ears) at their first and at their last examinations, which are shown in Fig. 2, A, so closely resemble the corresponding audiograms for the entire group of untreated children, given in Fig. 1, that only by careful inspection and comparison of the two sets of data can even minor differences be noted. I doubt that the minor differences have any significance.

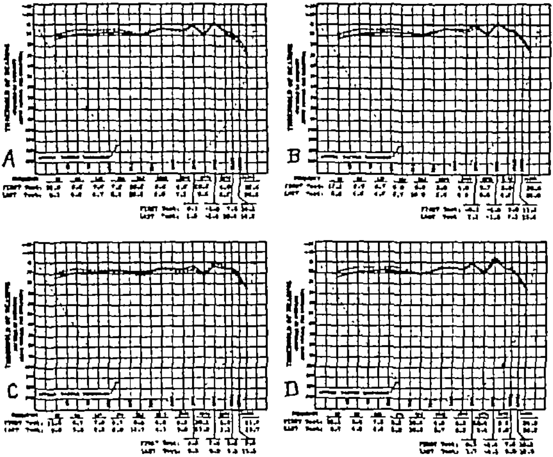

Fig. 2. The average changes in hearing acuity that occurred in each of the four subgroups of the 259 untreated children (see Fig. 1) on the basis of tubal orifice conditions at the times of the first and of the last examinations. See text matter for size of the subgroups, etc.

Sixty-three children, or about one-fourth of the total group of 259 untreated children, had normal appearing tubal orifices at both the first and the last examinations; in other words, they had, probably throughout the entire period of the study, what is usually regarded as the ideal, or the desired, condition of nasopharyngeal lymphoid tissue with respect to its possible effect on hearing acuity. On theoretical grounds it might well be argued that the baseline for normal age changes in hearing acuity of children should be limited to the data for this subgroup. To do so, however, would make no essential difference, as may be seen by comparison of the average audiograms for this subgroup of 63 children (126 ears), shown in Fig. 2, B, with the corresponding data for the children of the entire group (Fig. 1) and with the data for the children whose nasopharyngeal lymphoid tissue in the tubal orifice regions underwent spontaneous regression satisfactorily during the period of observation (Fig. 2, A).

Thirteen of the untreated children who had normal tubal orifices at the first examination had one or both tubal orifices abnormal in appearance, on nasopharyngoscopic inspection, at the time of the last examination; in other words, hyperplasia of lymphoid tissue had developed in the tubal orifice region during the period of the study. The average audiograms for this small subgroup of 13 children (26 ears) are shown in Fig. 2, C. The slight variations from the changes shown in the two preceding subgroups (cf. Fig. 2, A, and Fig. 2, B) are probably due in large part to the small number of cases in this subgroup; certainly the development of lymphoid tissue in the tubal orifice regions did not affect the acuity of hearing for high tones unfavorably.

The average audiograms for the fourth subgroup of the 259 untreated children, made on the basis of tubal orifice conditions, are shown in Fig. 2, D. The subgroup consists of 53 children whose tubal orifices, one or both, were abnormal in appearance at the times of both the first and the last examinations. The shortest interval between the first and the last examinations was 6 years, the average interval was slightly over 6½ years. Presumably an excess amount of lymphoid tissue was present in the tubal orifice region at least most of the time during the interval; this was true for the children who did have intermediate examinations made during the first two years of study. On theoretical grounds these children, who had persistently abnormal tubal orifices for over six years each, should have suffered a greater average loss of hearing acuity than did the children of the subgroups in which spontaneous regression of lymphoid tissue occurred (Fig. 2, A) or in which the tubal orifices were normal throughout the period of observation (Fig. 2, B). Actually, such a loss of thresholds did not occur. Comparison of the data in part D of Fig. 2 with that in parts A and B shows that the gain that occurred in the average thresholds for low tones is strikingly similar to that which occurred in the other two large subgroups, and, most surprising of all, that this is the only one of the subgroups in which a slight loss in acuity of hearing for the very high tones did not occur. I do not know whether the difference in the changes of thresholds for high tones between this and the other subgroups is statistically significant in the sense of indicating that persistent lymphoid tissue in the tubal orifice region has a beneficial effect on hearing acuity for high tones, but I am certain that the subgroup is large enough — 53 children (106 ears), or slightly over one-fifth of the entire group — that the data prove conclusively that the lymphoid tissue persistently present in the tubal orifice regions of these children for over 6 years did not exert a deleterious effect upon hearing acuity during the period of the study.

CHILDREN WITH IMPAIRED HEARING; TREATED.

With the above data about the untreated children as a background, let us turn now to a consideration of the changes in hearing acuity that occurred in the children who had impaired hearing at the time of their first examinations and who were treated, early in the course of the study, by means of irradiation of nasopharyngeal lymphoid tissue, or by, in a few instances, some combination of throat surgery and nasopharyngeal irradiation, or by, in cases of suppurative otitis media, some form of local non-surgical therapy in addition to irradiation of the nasopharynx.

All the treated children who returned in 1946 or 1947 were among those seen repeatedly during the course of the first two years of the study and whose condition at that time was reported in 1942. In all but ten per cent of the treated children success had been attained in shrinking the nasopharyngeal lymphoid tissue before the end of the two-year period of repeated re-examinations, usually long before.[9] By the time of their return in 1946 or 1947 the treated children, almost without exception, had both tubal orifices entirely normal in appearance, on nasopharyngoscopic inspection. This being the case, the report on the changes in hearing acuity that occurred in them does not have to be complicated by subdivisions of groups on the basis of tubal conditions, but the material can be grouped entirely on the basis of type of hearing impairment present at the time of the first examinations, before treatment was started.

HEARING IMPAIRMENTS LIMITED TO HIGH TONES.

The largest group of the treated children is that in which some form of impairment of hearing for high tones existed, the hearing for low tones being good, usually up to 2048 or higher. The average audiograms for the entire group are presented in Fig. 3. The code is the same as that used in the charts about the untreated children (Figs. 1 and 2); the solid line represents the arithmetical averages of the thresholds at the first examination, the dotted line the corresponding data for the last examination. Each of the lines in this chart shows the average of the thresholds of 95 ears. The designation for this group is given as ears, instead of as children, because some of the impairments were unilateral; it would have confused the determination of the effect of nasopharyngeal irradiation on this form of hearing impairment to have included in the calculations the opposite ears, some of which had good hearing and some of which had impaired hearing for all tones. Comparison of Figs. 1 and 3 shows that the baselines, *i.e.*, the averages of their first audiograms, differ for the high tones in the untreated and the treated groups in the manner that would be expected from the basis used for the selection of children for treatment, but that the average *changes* in the acuity of hearing that occurred in the two groups during the period of observation resemble each other closely. The treated group, like the untreated, had a slight improvement in thresholds for tones of the low range of frequencies and a slight

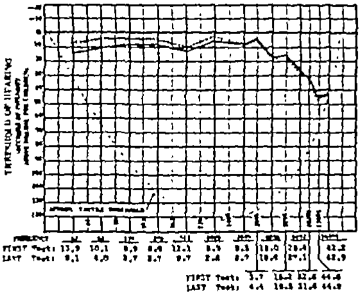

Fig. 3. The averages of the first and of the last audiograms of 95 ears of children who had some form of impaired hearing for high tones and who were treated by nasopharyngeal irradiation. The interval between the tests was six years or longer in each case.

additional impairment for high tones. The only real difference in the *changes* that occurred in the average acuity of hearing of the two groups is that the improvement at the low frequency end of the scale includes, in the treated group, the tones of the frequencies of 512 and of 1024 cycles, as well as the ones from 256 downwards. The average changes for the high tones are all slight in amount in the treated group, and the only one for which the average threshold improved at all, from 2048 cycles upwards, was 10321. The average gain at this frequency amounted to only one decibel.

It is well recognized that a hearing impairment limited to high tones may be of the so-called "gradual" type or of the so-called "abrupt" type. The differentiation, which is not always clearcut, is usually made nowadays on the basis of the appearance of the threshold audiogram.[10] To determine whether the effect of nasopharyngeal irradiation may differ with the type of high-tone loss, the material used for Fig. 3 has been subdivided.

The "gradual" high-tone losses. Fig. 4, A, presents the data for the 43 ears that had, individually considered, an impairment of the "gradual" type. Even a casual inspection of the charted data shows that in this subgroup a slight gain in average hearing acuity did occur for three of the high tones, those with the frequencies of 8192, 10321 and 13004 cycles per second. A closer scrutiny of the individual records of this subgroup, made in an attempt to learn to what factor the

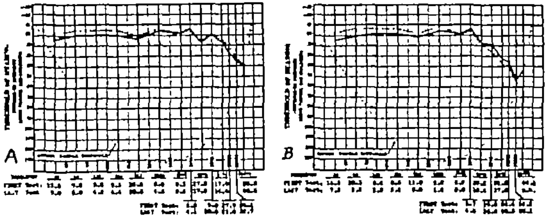

Fig. 4. The high-tone loss cases (see Fig. 3) subdivided on the basis of "gradual" or of "abrupt" type loss at the first test.

improvement might be attributed, revealed that only 12 of the 43 ears had an average change, for the four highest frequencies tested (8192 upwards), of more than 10 decibels; nine had become better, three had become worse. Seven of the nine that definitely improved had had a return to normal or essentially normal thresholds for all of the high tones except 16384. It is of interest to note, also, that the three ears for which hearing acuity definitely became worse all had a change in type of high-tone loss, from a "gradual" to an "abrupt" form of impairment; none of the three had undergone a progression of the "gradual" type of loss present at the first examination. Nothing has been found, in the form of relationships to other items about the condition of these children, to explain why some ears of this subgroup had definite changes in hearing acuity while the majority did not.

The "abrupt" high-tone losses. Fig. 4, B, presents the data for the 52 ears that had, at the first examination, some form or other of an "abrupt" type of hearing loss for high tones. Inspection of the charted averages of thresholds shows that in this subgroup, as in that of the ears with a "gradual" type of high-tone loss, the improvement for tones at the lower end of the scale includes the frequencies of 512 and of 1024 cycles, but that, in contrast to the other subgroup, hearing acuity for all the high tones has on the average become worse. In order to ascertain whether the progressive impairment for high tones, for the subgroup as a whole, was associated more with one subtype of "abrupt" loss than with another, the material on which Fig. 4, B, is based has been further subdivided on the basis of how many of the high tones were poorly heard at the first examination.[10] This subdivision of necessity makes the number of ears in each category smaller than is desirable for significance. For whatever they may be worth in indicating trends, however, the averages for the categories that contain 6 or more ears each are presented in Fig. 5, parts A to D. Fig. 5, A, shows the averages of the first and of the last audigrams of 20 ears, each of which had, at the first examination, good hearing for tones up to and including the frequency of 10321 cycles, and markedly im-

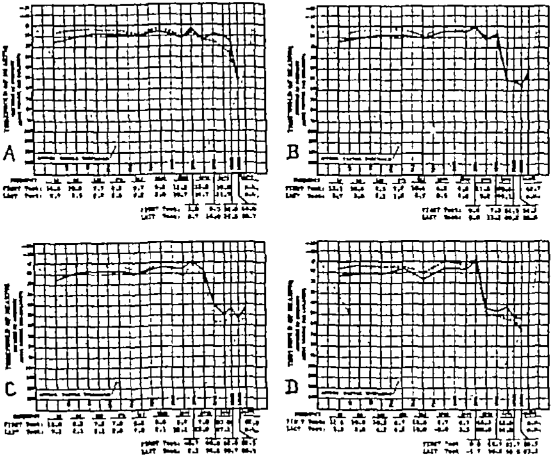

Fig. 5. Subdivision of the "abrupt" type loss cases (see Fig. 4B) on the basis of how many of the high tones were poorly heard at the time of the first test. See text matter for size of groups, etc.

paired hearing, or a total loss, for the two highest tones tested, 13004 and 16384; Fig. 5, B, gives the corresponding data for the 18 ears that originally had the frequency of 5793 cycles as the highest one well heard; Fig. 5, C, gives the data for the 7 ears that at the first examination had markedly impaired hearing for the five frequencies tested above that of 4096 cycles; and Fig. 5, D, gives the data for the 6 ears with which 4096 and all higher frequencies were poorly heard at the beginning of the study.

The progression of the impairment of hearing for high tones that took place in the ears with "abrupt" type losses, as indicated by the observations just presented, occurred in spite of the successful shrinkage, by means of nasopharyngeal irradiation, of lymphoid tissue in the regions of the tubal orifices of these ears. Another item of interest today is that in not even one instance in the entire subgroup of 52 ears was the highest tone that was well heard of a higher frequency at the last examination than at the first examination. These two facts, in spite of the small size of the categories of the subgroup, afford strong support to the view that the cause of "abrupt" type high-tone losses of hearing in children is not related to the presence or absence of lymphoid tissue in the nasopharynx.[8]

MEDERATE IMPAIRMENT OF HEARING FOR ALL TONES.

Seventeen of the children who returned for re-examination in 1946 or 1947 had had, at the time of their first examinations, a moderate impairment of hearing for all tones; *i.e.*, the thresholds for most tones were in the 25 to 40 decibel zone. In seven of the children the impairment for all tones was bilateral, in ten it was unilateral; thus the total group with impaired hearing of this type consists of only 24 ears.

Previous to their first examination in the present study, all but three of the children in this group had had a suppurative otitis media, which history was confirmed in each case by the appearance of the tympanic membrane, and 12 of the 17 children had had a tonsillectomy and adenoidectomy performed. All of the children were treated by nasopharyngeal irradiation, using radon in a brass applicator with a wall thickness of 1 mm. The usual dosage given at a treatment with this applicator was the equivalent of 2.2 gram minutes of radium, to each side of the nasopharynx. The average number of treatments given the children of this group was 4½; two boys received 8 treatments each. The total irradiation received by each side of the nasopharynx of these two boys was, respectively, 18.7 gram-minutes equivalent in 15 months, and 19.3 gram-minutes equivalent in 21 months. In addition to the irradiation treatments, several of the children had local treatments of the ears, at the times of suppurative episodes, and 4 of the 5 children who had not had a tonsillectomy and adenoidectomy previously had this operation performed during the period of observation. Operation was strongly advised for the fifth child also, but permission for operation was refused by the parents. In other words, treatment of the children of this group was not limited to nasopharyngeal irradiation, but efforts were made to improve the hearing by whatever treatment procedure seemed advisable.

On their return in 1946 or 1947, all but one of the 17 children in this small group had normal appearing tubal orifices; this good result includes the boy for whom operative permission was refused. He did have a total of six irradiation treatments during a one-year period of time. Three of the children had, at the time of their return, a suppurating ear.

The average of the audiograms of the 24 ears in this group, at the times of the first and of the last examinations, are presented in Fig. 6. Inspection of the charted data shows that on the average these ears had a real improvement in hearing acuity during the period covered by the study. Inspection of the individual records shows that none of the ears became markedly worse, that several of them had only slight improvements, and that about half of the ears had marked degrees of improvement. In Fig. 7 are presented the first and

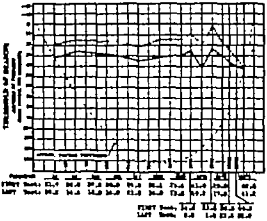

Fig. 6. The averages of the thresholds at the first and at the last tests of the group of 24 ears that had, when first examined (solid line) moderate impairments of hearing for all tones. All had nasopharyngeal irradiation, some had other treatment also. See text matter.

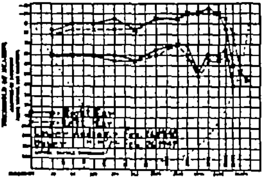

Fig. 7. The actual audiograms (not averages, as in the other figures of this paper) of the child whose hearing improved the most of any during the period of the study

olds in the two groups of children warrants the conclusion that the nasopharyngeal irradiation treatments received by the children with impaired hearing for high tones had no effect on their acuity of hearing, either beneficial or otherwise. The question of why the baselines of the two groups, for high tones, were different at the beginning of the period of observation falls outside the province of this Symposium.

For the small group of children who originally had impairments of thresholds for all tones, the audiograms of the first and of the last examinations show that on the average a considerable improvement in hearing acuity occurred during the period of observation. Whether or not the improvement was a result of the treatments received is open to question. Inspection of the individual records of the children in this group reveals a lack of homogeneity of important factors that is in marked contrast to the similarity of the individual records of the children of the groups with various forms of impaired hearing for high tones only. Furthermore, most of the average improvement shown in the audiogram is attributable to the inclusion in this group of three children whose hearing, bilaterally, returned to essentially normal. Such improvements in hearing are well known to occur spontaneously sometimes in children who have moderate impairments of hearing for all tones. A suitable control group of untreated children, followed for a similar period of years, is not available for evaluation of the factor of spontaneous improvement of hearing. For the above reasons I believe that the follow-up study reported today does not contain enough evidence to be convincing with respect to the effect of nasopharyngeal irradiation on the hearing acuity of children who have impairments of thresholds for all tones.

In connection with the above discussed group, it is of interest to note that only 38 of the 1365 children examined in the original survey during the school year 1939-1940 had impaired hearing for all tones, either with one or with both ears, and that 30 of these 38 children either had previously had, or had at the time of the first examination, a suppurative otitis media.[12] The well recognized importance of suppurative otitis media as a cause of handicapping degrees of impaired hearing is confirmed by the observations, but the data of the present study afford no evidence as to whether or not the incidence of ear infections in early childhood is actually reduced by nasopharyngeal irradiation.

The changes that occurred in the hearing thresholds of the group of 259 untreated children are in some respects the most interesting of the findings made in this follow-up study. All of the children of this group had at least fairly good hearing at the time of their first examinations and none of them had any treatment of ears, nose or throat during the period of observation. The changes that occurred may therefore properly be regarded as normal for the age period covered. The fact that, for the group as a whole, a slight loss occurred in the thresholds for very high tones, is in good agreement with what was to be expected from all the studies that have been made of normal age changes in adults.[1,2,3,11,14,16] The studies of adults, however, have given no reason to expect that an improvement in hearing acuity for low tones occurs during the childhood years. This observation therefore requires further consideration. Three items favor the view that the recorded changes in thresholds actually represent a gain in hearing acuity for low tones. The items are: (1) the care that was taken throughout the study to maintain uniform, and proper, conditions for the making of hearing tests; (2) the fact that all the subgroupings that have been made of the material, including ones that have not been presented today, show similar changes in the average thresholds for the low tones (32, 64, 128 and 256 cycles) during the period of observation; and (3) the likelihood that any so-called "practice effect" would have been operative for the responses to all of the tones used in the tests, not limited to the responses made to tones of one range only.

I believe, but have no evidence to support the hypothesis, that the above recorded "normal" improvement in thresholds for low tones is the result of a purely physical phenomenon, most probably a change in acoustic impedance related in some way to growth changes in the average size of the middle ear cavity and the external auditory canal. I doubt that the improvement in hearing which appears to occur normally during childhood, for low tones, results from any organic changes in the inner ear or in the auditory pathways of the brain. Certainly the observations reported today rule out spontaneous regression of lymphoid tissue in the region of the nasopharyngeal orifice of the Eustachian tube as the remote cause of the improvement under discussion, which occurred also in the group of children in whom spontaneous regression did not occur and in the group that had normal appearing tubal orifices throughout the period of observation.

The average change in hearing acuity for low tones that occurred in the children is so small that it would not be considered significant in comparing tests of an individual, but it occurred so consistently in the groupings of the material of the present study that I believe it to be a fundamental phenomenon in the age changes related to hearing; certainly it should be looked for by others who have material suitable for the purpose.

SUMMARY.

The title of today's Symposium stresses the irradiation aspect of the basic topic, which is lymphoid tissue in the nasopharynx. The data of the follow-up study of school children reported above yield information about the broad aspect of the relation of nasopharyngeal lymphoid tissue to hearing acuity as well as information about the effect on hearing thresholds of treatment of this tissue by irradiation.

The shortest interval between the first and the last hearing tests of the children in this study was six years, the average interval was slightly over 6½ years, both for the treated and for the untreated children. The average age of the children at the time of the first examination was 9½ years; the age range at that time was from 8 to 13 years.

In a group of 259 children (518 ears) whose hearing was good at the first examination and who had no throat treatment, either irradiative or operative, during the entire period of the study, a slight average gain in hearing acuity occurred for low tones, as measured by audiometric air-conduction thresholds, and on the average a slight loss occurred for high tones. The gain for low tones occurred consistently in each of the subgroups based on the four possible combinations of normal or of abnormal tubal orifices at the beginning and at the end, respectively, of the period of observation. One subgroup, however, had on the average no loss for high tones; it consists of 53 children (106 ears) whose tubal orifices were abnormal in appearance, on nasopharyngoscopic inspection, at both the first and the last examinations. The greatest average loss for high tones occurred in the subgroup of 68 children who had normal appearing tubal orifices at the beginning and at the end of the study.

A group of 95 ears of children who had impaired hearing only for high tones, either bilaterally or unilaterally, and who were treated by irradiation of the nasopharyngeal lymphoid tissue, showed an average gain for low tones similar to that in the untreated children, in whom it is regarded as normal for the age period, and a slight additional loss for high tones. *In no instance of the 52 ears that had an "abrupt" type of* high-tone loss was the highest tone that was well heard of a higher frequency at the last examination than it was at the first examination.

A group of 24 ears that had a moderate impairment of hearing for all tones — "flat" audiograms at the 25 to 40 db. level — had, as a group average, real improvements in hearing acuity for all tones, after irradiation of the nasopharynx. About half of them, however, showed either very little or no improvement in hearing acuity.

The broad conclusion is reached that previous ideas, widely prevalent, of the effect of nasopharyngeal lymphoid tissue on hearing and of the effect of nasopharyngeal irradiation on impaired hearing, need to be revised.

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OBSERVATIONS THROUGH COCHLEAR FENESTRA.*†

H. B. PERLMAN, M.D.,
Chicago, Ill.

The development of our concepts of the physical phenomena taking place in the cochlea during acoustic stimulation has been largely dependent upon the study of the anatomy of the cochlea, particularly as observed in microscopic sections. The bioelectric and psychological phenomena have also been used as a base for our interpretations of the mechanics of cochlear function. In spite of a tremendous amount of thought and effort dating back to De Verney, in 1683, the separation of the purely physical from the neural phenomena in the cochlea remains obscure. The solution of this problem is necessary for an orderly development of our understanding of the function of this small complicated peripheral acoustic receptor.

Great difficulty in direct inspection of the cochlea during acoustic stimulation has also led to the use of models for clarifying the physical processes that may occur within the cochlea in response to acoustic stimuli. Models have been constructed on conceived rather than measured physical properties of the cochlea. Gradually, as the result of all these varied observations, some form of frequency selectivity in the cochlea has been agreed upon by most observers.

Direct observations of the cochlea during acoustic stimulation have been strikingly absent from the extensive background of anatomical and physiological studies upon which our concepts of cochlear function are based. We are indebted to Békésy[1,2] for working out the methodology for making direct observations on the cochlea during static and acoustic

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*This work was done in part under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

†From the Division of Otolaryngology of the University of Chicago.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 12, 1949.

pressure stimulation and for contributing most of the known facts about the physical properties of the cochlea and its reaction to acoustic stimulation. Békésy, using new techniques, during the past decade has presented a great deal of new fundamental information about the physical responses of the cochlea. He first made direct observations on the volume elasticity of the basilar membrane and the cochlear duct and found that the elastic component of the mechanical impedance of the basilar membrane varied from apex to base by a factor of about 100 to one.

The above statement, using the term mechanical impedance, needs some clarification for the clinician.

Any structure will offer mechanical impedance to vibration. Such impedance is due to three fundamental properties: mass, elasticity and friction. When a driving force acts to set a structure into vibration, forces which tend to resist the motion result from these three fundamental properties which are, respectively:

1. An inertia force due to the mass.
2. A stiffness due to elasticity.
3. A resistance force due to friction.

The effects of these three properties are combined mathematically into a single quantity, known as the mechanical impedance of the structure to vibration. The amplitude of vibration is directly proportional to the amplitude of the driving force but inversely proportional to the mechanical impedance.

At every point along the cochlea, the structures of the cochlear duct offer a form of mechanical impedance to the driving forces in the perilymph. All but one of the structures of the cochlear duct yet studied exhibit relatively constant impedance throughout the entire length. The basilar membrane, however, has been found by Békésy to have a varying impedance along its length, its impedance being highest at the base and lowest at the apex. That is, the amplitude of displacement of the basilar membrane under static pressure varies by a factor of 100 to one from apex to base. This is a measure of the elastic component of impedance.

Békésy observed that the shape of the depression of the basilar membrane to a point source does not indicate that the membrane is under any special transverse tension, but suggests rather that the membrane responds as an unstressed gelatinous plate with varying volume elasticity. Using an artificial stapes so as to maintain a constant amplitude or volume displacement of the stimulus, he studied through suitable windows the nature of the vibrations induced and found that there was a region of maximum amplitude of vibration of the cochlear duct. This region changed with the frequency of the stimulus. He found,[3] probably for the first time, a definite frequency distribution along the basilar membrane for frequencies between 25 and 2000 cycles per second. He then determined the pattern of response at a given position on the membrane for varying frequencies with constant amplitude and volume displacement of the stapes. This indicated that from a given point along the basilar membrane there was a rather wide flat frequency response instead of a sharply peaked response to a very narrow frequency band. The frequency response was that of a considerably damped vibration. The high damping was corroborated by other ingenious methods of observation, *i.e.*, using a rotating objective in the microscope, and was measured.[4]

The apical end of the cochlear duct was seen to vibrate maximally to very low frequencies and to stop vibrating as the frequency of stimulation was raised. Békésy[5] has already measured the volume displacement of the round window for sounds of constant pressure at the drum and found that the volume displacement of the round window was rather uniform for frequencies up to about 3000 cycles. For a large sound pressure this volume displacement was about one-millionth of a cubic centimeter. The amplitude of the movement of the cochlear partition for this stimulus was about one-thousandth of a millimeter. For acoustic stimuli of constant amplitude the basilar membrane, therefore, moved less for low frequencies, since a longer segment of the membrane was set into vibration than for high frequencies where the apical end was by-passed and no longer vibrated. For a constant volume displacement of the stapes the amplitude of vibration of the basilar membrane to higher frequencies was increased only by a factor of 10 to one, and while it was in the right direction to explain the increased sensitivity of the ear at high frequencies over the low frequencies, the increase was of much smaller amplitude than that observed in the human auditory threshold. Physical laws indicated that the continuously varying mechanical impedance of the basilar membrane would result in a continuously varying velocity of the wave of deformation as initiated by the moving footplate. He measured,[4] for various positions near the apical end of the cochlea, the velocity of propagation of a sharp pulse. A sharp pulse initiated at the stapes arrived 1/1000 second later 25 mm. away on the basilar membrane. This was the region responding maximally to 500 cycles per second. By a remarkable experiment[6] he found that the movement of the basilar membrane is in phase with that of the stapes only at very low frequencies and as the frequency is increased there is an ever greater phase lag between footplate movement and movement of basilar membrane. This indicated that the stimulus is propagated as a traveling wave, becoming shorter as the frequency is increased; however, all these stimuli travel along the basilar membrane with varying velocity, being rapid at the narrow end and slowing down as they reach the region of maximum response. Beyond this region the disturbance rather rapidly dies out.

The conducting mechanism has long been known to be acoustically active in the fresh cadaver and even when the fresh temporal bone is removed from the cadaver and kept in a cold, moist chamber. Many important physical properties of this mechanism in response to sound have been determined with such preparations. The state of the tissues and fluids within the labyrinth during acoustic stimulation has been more obscure, but with fresh and properly preserved specimens it can easily be shown that many of the structures seen under the microscope in temporal bone sections can be visualized in action in the fresh state. This has been clearly demonstrated to the clinician in the living human ear where the membranous semicircular canal and ampulla can be identified and manipulated during fenestration and even partially evulsed as in the surgical treatment of Ménière's disease. Similar observations in the fresh human cadaver preparation reveal that these parts of the membranous labyrinth are also easily demonstrable. In these preparations the fluid in the labyrinth is preserved and the membranous canal can be stretched and partly evulsed as in the living ear. It can be freed from its attachment to the bony canal, compressed and moved about and perforated. Some conception of the physical character of the endolymph can be obtained.

The modern methods of exposure of the labyrinth spaces under a dissecting microscope, using dental drills of various degrees of size and fineness with irrigation of the area to remove bone dust, has been highly developed by Békésy and permits the creation of clean fistulas in these specimens opening into almost any part of the labyrinth, including the various coils of the cochlea. The use of normal saline or Ringer's

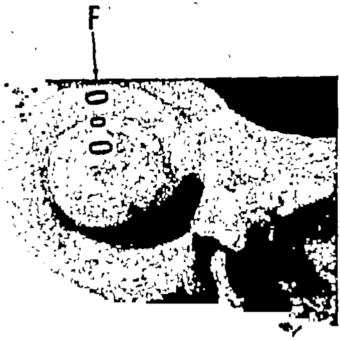

Fig. 1. Dissection of the bony cochlear capsule indicating the approximate size and location of the fenestra (F) that can be made looking into the scala vestibuli of the apical, middle and basal coil while still preserving the conducting machinism for transmission of the acoustic stimulus.

solution for irrigation is useful to prevent trauma to the membranous wall of the cochlear duct due to osmotic tension differences. Using a binocular microscope, usually with a 30 to 70x magnification, or a water immersion lens for magnification up to 150x, the structures within the cochlea are made available for study.

The petrous pyramid is cleaned and the tensor tympani muscle is partially removed with its bony partition so that the area of the apex and middle coil of the cochlea can be exposed while still preserving the conducting mechanism. Some of the basal coil can also be exposed in this way from the scala vestibuli side (see Figs. 1, 2, 3). As Békésy has shown, exposure of the basal coil nearer to the vestibule can be obtained by drilling out the internal acoustic meatus and opening the scala tympani of the basal coil for a view of the

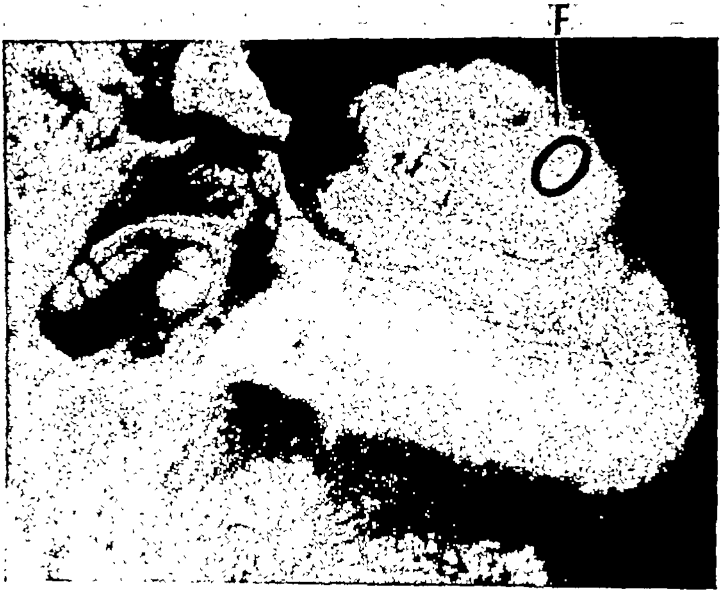

Fig. 2. Cochlear capsule as viewed from the labyrinth windows. Oval area indicates approximate size of observations window (F) used in these experiments.

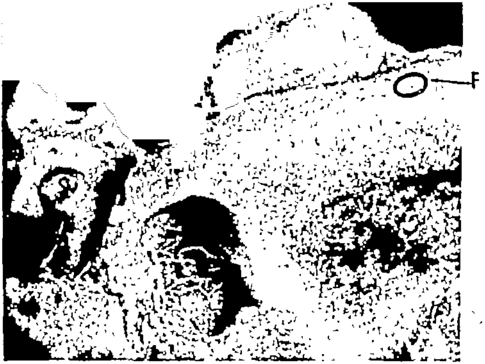

Fig. 1. View of round window area (RW) of the cochlea in a human fetus at birth with the beginning of the basal coil in focus. Oval area indicates the approximate size of observation windows (F) used. The head of the stapes is indicated by (S).

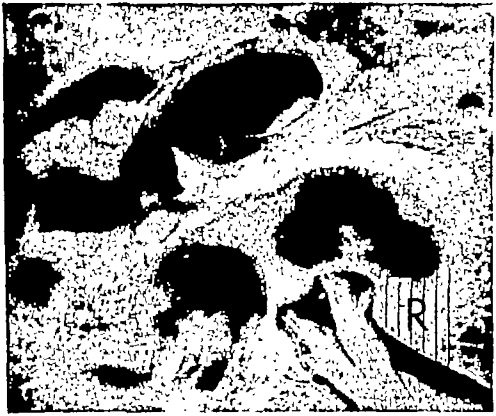

Fig. 4. Cross-section of the cochlea indicating the relative position of the bone removed (R) at the internal meatus for the creation of an observation window looking into the scala tympani of the basal coil close to the vestibule. The spiral ligament, basilar membrane and spiral lamina of the basal coil are clearly seen.

basilar membrane and spiral lamina (see Figs. 4, 5). In this manner the cochlear structures can be visualized and the fluid within the cochlea can be preserved. Through these windows it is easy to recognize the Reissner's membrane, spiral ligament and spiral lamina. Reissner's membrane and other membranous structures can be more clearly delimited and observed during acoustic movement by placing specially prepared highly reflecting silver crystals in the perilymph and allowing them to settle and adhere to the membranous and bony structures.

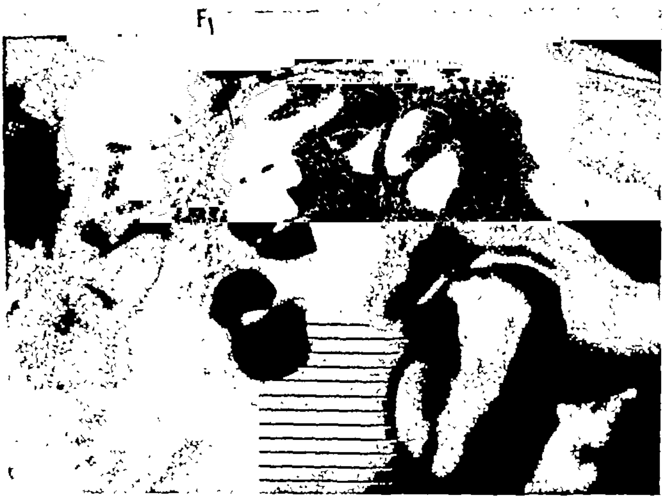

Fig. 5. Cross-section of the cochlea to indicate the position of the observation windows (F) relative to the cochlear duct. Reissner's membrane is clearly seen. Shaded area represents capsule bone removed at the internal meatus to expose the proximal end of the basilar membrane.

The instruments needed to manipulate these cochlear structures in the fenestra consist of various sized hairs fixed to the end of a wooden applicator or glass rod. Fine glass, platinum or palladium probes can also be made in this way. Steel needles, straight and shaped into fine hooks, are used as in a fenestration operation to remove bone fragments from the edge of the fistula to open the endosteum and remove fragments lying on the cochlear duct, etc.

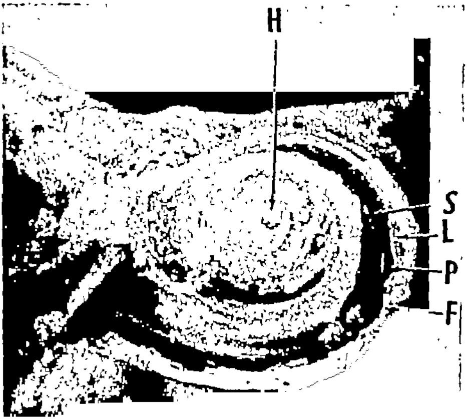

Fig. 6. Widely opened cochlea to show the relation of the scalae, the bony spiral lamina and basilar membrane. Oval area indicates approximate size of field exposed by fenestra (F). A white line (P) is placed on the basilar membrane above fenestra to indicate relative thickness of hair probe used through the fenestra to depress the basilar membrane and manipulate Reissner's and the tectorial membrane. An arrow points to the spiral ligament (L) detached from the cochlear capsule and another arrow points to the edge of the bony spiral lamina (S) to which the basilar membrane is attached. The helicotrema can be made out at (H).

It is easy to tear Reissner's membrane and thus to obtain a direct view of the basilar membrane and its superimposed structures. While the details of the organ of Corti cannot be made out, the tectorial membrane is easily visualized as a flat ribbon of gelatinous material near the free lip of the bony spiral lamina and appears to be adherent to the cells on the basilar membrane below it (see Fig. 5).

The tectorial membrane can be lifted off these cells and a long ribbon of tectorial membrane can be freed. This tectorial membrane has considerable resistance to tearing, coming away from a large area. It can even be delivered out of the observation window before the fine hook or needle succeeds in breaking it as it is lifted off the basilar membrane. As Békésy[4] has observed, point pressure on the tectorial membrane produces an elongated hollow depression in contrast to the other surfaces that exhibit a circular depression. The tectorial membrane exhibits a different elasticity in the transverse direction than it does lengthwise. In the lengthwise direction it is more stiff. According to Békésy, the reduced stiffness in the transverse direction, peculiar to the tectorial membrane, permits a pressure drop along the cochlear duct that favors frequency analysis.

The basilar membrane can be depressed with a fine human hair or fine metal rod, and a judgment of its volume displacement or the elastic component of its mechanical impedance at various positions along the cochlea can be determined. The limits of its width are easily seen as it bridges across as an almost transparent sheet from the spiral bony lamina to the spiral ligament (see Fig. 6). The shape of the deformation of the basilar membrane to a point pressure indicates that it has no special transverse tension as compared to its longitudinal tension. The depression is shallow and circular through most of its length. The basilar membrane may also be perforated with either a human hair or a fine wire. The shape of the hole so produced through most of the basilar membrane is circular and again indicates that it is exposed to equal forces in the transverse and longitudinal direction. On the basis of this response to pressure by a point source, Békésy likens the basilar membrane to an unstressed gelatinous plate. Using hairs of graduated and measured stiffness and by using a constant pressure of 1 cm. water, Békésy has obtained quantitative judgment of the elasticity or stiffness of the basilar membrane. The membrane is exposed to restoring forces since it returns from its displaced position when depressed with a probe. Békésy showed that movement of the cochlear partition imparted by acoustic stimuli dies out rapidly, indicating considerable damping. The degree of stiffness of the basilar membrane is easily seen to vary along the cochlear duct when observing the amount of deformation to a probe of suitable stiffness. The membrane is more stiff in its narrow basilar portion and relatively more movable at its apical or wide end, where, indeed, the modiolus and bony lamina can also be moved but with greater forces. At this end the helicotrema can be clearly visualized with its terminal portion of the cochlear duct (see Fig. 7). This area is rather difficult to expose without tearing the Reissner's membrane or fracturing the modiolus. Békésy found a variation from the wide apical end to the narrow basal end of 100 to one in volume displacement of the basilar membrane to a constant static pressure of 1 cm. water.

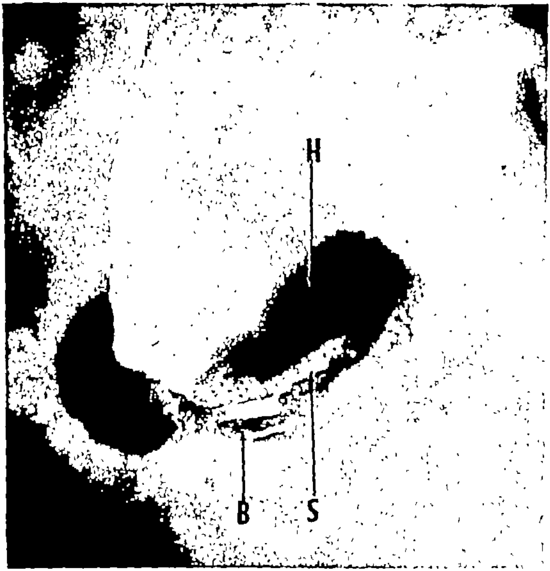

Fig. 7. Dissection indicating the portion of the helicotrema (H) relative to the end of the bony spiral lamina (S). The basilar membrane is indicated at (B). The thickness of the arrow corresponds to that of the fine probes used to depress the basilar membrane or manipulate Reissner's and the tectorial membrane (not shown in the illustration).

The spiral ligament can be observed as it is moved by a hair probe and it can be easily peeled from its attachment to the outer bony cochlear wall (see Fig. 6). The ease with which this structure can be peeled from the outer wall and the retention of its form and that of the basilar membrane, even when so freed by the cochlear fluids, indicate that no real ligamentous pull on the basilar membrane is carried out by the spiral ligament.

The appearance and the marked resistance of the bony spiral lamina to pressure with a fine hair easily defines the medial limit of the basilar membrane. The medial edge of the basilar membrane can also be seen as the location of the tectorial membrane.

The bony lamina with the rest of the modiolus can be inspected in the area exposed by the fenestra.

The greatly reduced resistance of Reissner's membrane to deformation with a probe is easily seen, also its rather uniform degree of resistance all along the cochlear duct. The flat 0.04 mm. wide silver crystals are excellent light reflectors and help to visualize more quantitatively the degree of movement of Reissner's membrane when touched by a hair probe or when moved by sound stimuli. There and elsewhere in the cochlea the change of the point of light into a line or oval indicates the degree and character of the resultant vibration. The fluid contents of the cochlear duct are qualitatively seen to resemble that of the perilymph. This is in marked contrast to the tectorial membrane that is seen as a thin gelatinous ribbon adhering to the cells on the basilar membrane.

The response of these various identifiable and visible structures of the cochlea to acoustic stimuli reaching it through the intact conducting mechanism can be observed by so preparing and imbedding the specimen that the conducting mechanism is preserved along with the external canal and only the cochlear area is exposed for fenestration.

Strong light reflected by a small mirror mounted on the microscope and focused to a fine point is needed to illuminate properly the structures within the 2 mm. fistula, using up to 70x magnification with the stereomicroscope. A water immersion lens with the apex of the petrous pyramid immersed in saline may be used for higher magnification while protecting the middle ear mechanism from filling with fluid during acoustic stimulation. The illumination of an "ultra pack" can also be used here. For acoustic stimulation the external canal is coupled through tubing to the loud speaker driver unit activated by a Hewitt Packard audio-oscillator and suitable amplifier.

For greater facility in making manipulations and exposing the structures within the fistula, the plaster imbedded bone is mounted in a clamp with a universal joint so that movement in all directions is possible. The area about the binocular scope is surrounded by a platform for a more secure rest of the operator's arms. The scope is mounted directly to the table top. With practice and with the arms placed in the same position and the instruments grasped in about the same position on the handle, they can be brought into the small field, about 3 mm.^{2} to 6 mm.^{2}, rather consistently.

The temporal bones of other mammals (see Fig. 8) (guinea pig, cat, etc.) can be similarly prepared and investigated for static and acoustic characteristics of the cochlea (Békésy[7]). The temporal bone in the human is more difficult to work with because of the relatively great thickness of the cochlear capsule, but when the bones from a newborn are used, this is greatly facilitated. Most of our observations in the human were made on full-term fetal temporal bones with aerated middle ears. Since the conducting mechanism and the cochlea are quite fully developed at birth, satisfactory information can be obtained. The amount of capsule bone that must be removed is minimal in these specimens. Stroboscopic illumination with a strobotac as well as constant illumination may be used for making observations of acoustic responses. Silver crystals, bone dust and lamp black particles suspended in the cochlear fluids can be observed for evidence of the behavior of the fluids in the cochlea during acoustic stimulation.

With the experimental procedure described, the great amount of activity observed in the fluids and membranous structures within the cochlea during acoustic stimulation

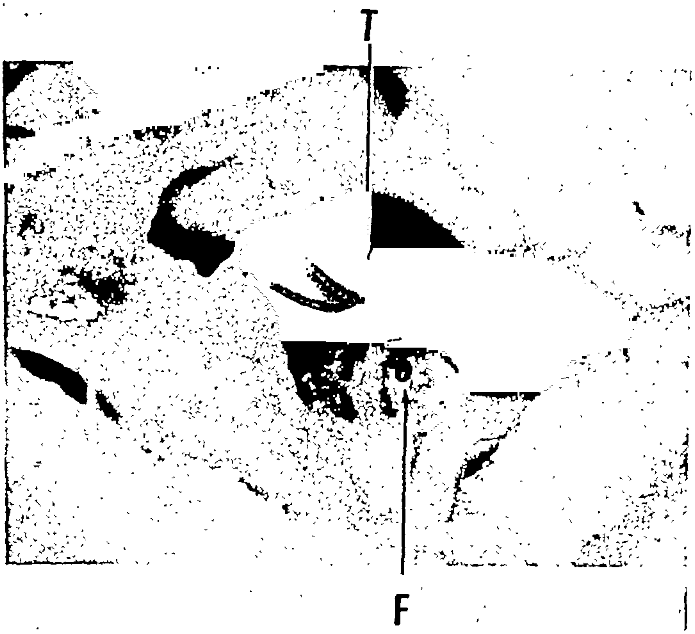

Fig. 8. Exposure of guinea pig ear with oval area indicating approximate size of fenestra (F) used for observing through the binocular scope, with about 60× magnification, the activity within the various coils of the cochlea during acoustic stimulation through the intact ossicular chain. The drum (T), malleus and incus are easily visualized just above the cochlea.

through the intact chain is striking. A knowledge of the behavior of fluids in motion (hydrodynamics) becomes at once of interest to the observer. For a clearer insight into the observed activity in the cochlea, one is led to a consideration of this relatively new and difficult field of physics. Known laws of fluid mechanics enter into the explanation of the visible activity such as eddy currents seen within the cochlea during acoustic stimulation. The particular structure of the organ of Corti itself, such as the pillars and fluid spaces about them, no doubt control the ultimate definition of the fluid movements that affect the end-organ.

It is possible to observe that the acoustic vibration of the cochlear partition in response to acoustic stimuli entering through the intact conducting mechanism is at right angles to the scalae and that Reissner's membrane moves with the basilar membrane. The cochlear duct appears to vibrate as a unit. At low frequencies (15 cycles per second), movement of the cochlear fluid on each side of the cochlear duct parallel to the scalae can be observed, as reflected by the action of particles suspended in it. As the frequency of the sound is increased, right angled movements of the cochlear partition are observed with greatest amplitude at the apex. As the frequency is further increased, above about 400 cycles, the apex is seen to come to rest and through windows in the middle and lower coil one sees active vibration of Reissner's membrane, etc. In these preparations this acoustic response can be observed in the basal coil up to about 2000 cycles in man and higher in the guinea pig. Transmission of these high frequencies is inefficient through the air in the coupled tubings between the speaker and the external canal. Movement of the fluid system consists in parallel to and fro pulsasations along the length of the cochlear duct for very low frequencies, eddy current parallel to the length of the cochlear duct exposed by the fistula, and eddy currents at right angles to the cochlear duct. These latter are very striking and can be made to shift with frequency and to change their velocity and radius with change of intensity. The velocity of the right angled eddy current is greatest as it moves past the basilar membrane and is least at the top of its rotation near the level of the observation window. These eddies may be small in diameter, being limited to the area immediately adjacent to the basilar or Reissner's membrane. They can be observed beyond the limits of the fenestra by tilting the preparation. The Reissner's membrane appears to move outward as a static displacement into the scala vestibuli while vibrating acoustically. This is true of the tectorial membrane while still attached and when partially loosened from the underlying cells. The static negative component in the response may be due to the asymmetry of the stimulus delivered by the chain to the cochlea. This stimulus is stronger on the negative side of center. The tectorial membrane can be torn off the underlying cells by large acoustic stimuli. Particles entering the cochlear duct through a tear in Reissner's membrane can be seen to execute eddy movement within the cochlear duct and at right angles to the basilar membrane. Eddy currents may be limited to the fluid near the cochlear duct or with larger stimuli may involve the fluid inward to the modiolus and up to the bony walls of the scala tympani or vestibuli at the observation window. The acoustic stimuli used to produce these movements are well within physiological limits. For stronger acoustic stimuli the lines of light produced by movement in one plane of a crystal attached to the Reissner's tectorial or basilar membrane can be seen to change into an ellipse, indicating that movement is taking place in more than one plane; that is, at right angles to the cochlear partition and parallel to it. The bony spiral lamina can also be made to oscillate in a complex manner with large stimuli while eddy currents in the fluid over the bony spiral lamina and modiolus are observed.

The frequency discrimination of the cochlea as displayed by the pattern of vibration of the membranous portions of the cochlear partition is a very broad one. The whole cochlear duct appears to be set in vibration by low frequencies while shorter and shorter segments are seen to be involved as the frequency of acoustic stimulation is raised. The wide extent of motion set up by low frequency sounds is in the proper direction to explain the broad masking produced by large low frequency sounds as compared to the narrowing of the masking limits with higher frequency of stimulation. Distinct cessation of vibration of the cochlear partition at the apical window is noted, while the middle coil window reveals very active movement at 400 cycles. Békésy found that for a constant volume displacement of the stapes, the cochlear partition moves with a smaller amplitude at low frequencies than at high frequencies. The variation in amplitude is about 10 to one. This is much smaller but in the direction of the movement of the drum necessary to elicit a threshold response. The drum moves over 1000 times as much at low frequencies at the threshold than at higher frequencies; hence, the threshold characteristics of hearing at low frequencies are not explained by the behavior of the cochlear vibration observed.

A proper judgment of the consistency and mechanical resistance of the basilar membrane to deformation as determined by direct observations upon it permits the construction of models to aid in understanding cochlear mechanics. From his studies in models we are further indebted to Békésy[3] for a clarification of some of the physical dimensions that are important in maintaining a frequency analysis along the basilar membrane.

A V-shaped slit between two razor blade edges inserted over a trough 2 sq. mm. in transverse area is considered by Békésy[3] as the most simple type of model of the cochlea (see Fig. 9). The separation of the razor blade edges approximates the varying width of the basilar membrane, and the space bridged by floating a thin rubber solution over it. This creates a very thin homogenous elastic plate upon which water is placed with silver crystals, lamp black and lycopodium to observe activity in the superimposed fluid and movement of the artificial basilar membrane. Sound is delivered by air conduction through a sealed-in large gauge hypodermic needle shoulder. Békésy has shown that such a simplified model works in a manner similar to that of a complete model with two fluid-containing chambers separated by the basilar mem-

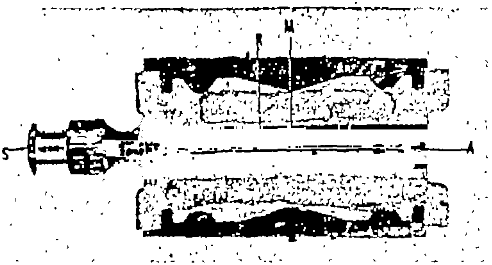

Fig. 9. Photograph of a simplified model of the cochlea, consisting of two razor back edges (R) across which a thin rubber solution is floated to make the basilar membrane (M). The acoustic stimulus is delivered through tubing from a loud speaker driven unit into the needle hub on the left (S). This opens into an air-containing trough (A) under the artificial basilar membrane. Fluid is placed over the membrane and silver, lycopodeum and lamp black particles used to observe the resulting motion.

brane. Responses up to 4500 cycles have been observed in this way with eddy currents at right angles to the membrane and limited to the fluid just above the membrane, instead of involving the entire overlying fluid. Changes in speed of motion in the eddy with respect to the artificial basilar membrane and increased velocity and diameter of the eddy as intensity of the stimulus is raised could be observed as in the cochlea itself as well as shifting of the position of the eddy along the membrane with changing frequency. The present concept of cochlear mechanics based on observations in models and in the cochlea as well as on some physiological experiments is that a wave of deformation of the cochlear partition is initiated by movement of the stapes and is propagated along the basilar membrane to reach a maximum at a position depending upon the frequency. This wave of deformation for all frequencies moves at varying velocity along the basilar membrane, being rapid at the beginning and slowing as it approaches the point of maximum response. The rate of propagation along the basilar membrane varies. For a position 25 mm. away from the narrow end, Békésy measured this velocity and found it to be 40 meters per second. The limits of variation in velocity throughout the entire length of the cochlear duct are now known. This velocity is, of course, much less than that of a sound wave in an infinite fluid medium (about 1500 meters per second and independent of frequency). The varying volume displacement of the basilar membrane along its length determines the rate of propagation and the region of maximum displacement. With higher frequencies the upper end of the cochlear duct is by-passed and the activity is seen to be confined to shorter and shorter segments of the cochlear duct.

The whole cochlear duct appears to vibrate as a unit at right angles to its length while the wave of deformation travels along the length of the duct. The column of fluid in the perilymph between the two windows does not move back and forth as a unit in phase with the motion of the footplate except at very low frequencies, *i.e.*, 15 cycles per second. Below 20 cycles the fluid moves through the helicotrema without affecting the cochlear partition. As the frequency is increased there is right angled movement of the partition and an ever greater phase lag between the movement of the stapes and that of the cochlear partition indicating a wave of motion being propagated along this partition. The distribution of amplitude along the basilar membrane for a given frequency is rather flat and does not provide evidence for a sharp definition of the stimulus; however, Békésy found that the phase of motion at a given position on the basilar membrane with respect to the motion of the stapes can be shifted by a very small change in frequency. Small disturbances in the pattern of vibration with change of frequency are manifested by this shift in phase. According to Békésy, the mechanical volume displacement of the partition together with the mass of the fluid very near to it effect its acoustic response, while the damping is a resultant of these forces and is quite large.

The rôle of the associated eddy current in effecting a pressure stimulation of the end-organ is considered in the mathematical analysis of the cochlea by Ranke[8] but minimized by Zwislocki.[9]

SUMMARY.

Direct measurement of some of the physical properties of the cochlea and observations of the cochlea during acoustic stimulation is possible with modern techniques and offers new important information about the physical processes in cochlear function.

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SUGGESTIONS FOR DETERMINING THE MOBILITY OF THE STAPES BY MEANS OF AN ENDOTOSCOPE FOR THE MIDDLE EAR.[*][†]

GEORG V. BÉKÉSY,
Cambridge, Mass.

Hearing losses are usually divided into two distinctly different categories: conductive deafness and nerve deafness. The treatments appropriate to these two defects are different. Although some patients fit neatly into one or the other of these two categories, many cases combine both nerve and conductive defects and are difficult to diagnose. For successful treatment of these combined cases it would be advantageous to know what percentage of the hearing loss is due to each of these two defects. This information might also help answer certain basic questions. For example, can a stapes fixation produce nerve deafness, or are stapes fixation and nerve deafness unrelated processes?

Because of the importance of this and similar questions, some means must be found for making precise diagnoses in combined cases.

DISTINGUISHING BETWEEN NERVE AND CONDUCTIVE DEAFNESS AT AUTOPSY.

It is possible to gain valuable information about the middle ear by making certain physical measurements prior to the histological examination of the cochlea. At autopsy it is possible to measure the conductive loss of a middle ear for the whole frequency range. In the event that it has been possible

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[*] This research was carried out under Contract N5ori-76 between Harvard University and the Office of Naval Research, U. S. Navy (Project Nr147-201, Report PNR-83). Reproduction for any purpose by the U. S. Government is permitted.

[†] Psycho-Acoustic Laboratory, Harvard University.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 15, 1949.

to obtain an audiogram before the death of the patient, we can subtract the values for conductive deafness, as measured at autopsy, from the overall deafness and thereby determine the extent of the nerve deafness. Having separated the values for conductive deafness and nerve deafness, we can then correlate them with the histological findings in the middle ear and in the cochlea. This makes the histological findings meaningful in terms of precise functional measurements.

The equipment used to measure conductive loss postmortem is illustrated in Fig. 1. The temporal bone is opened near the

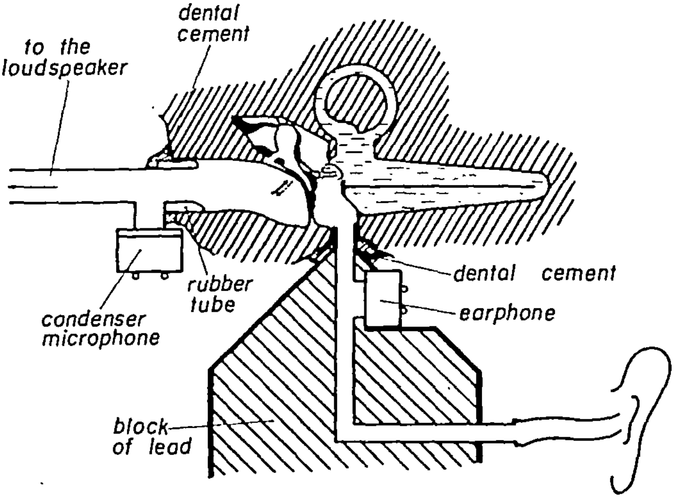

Fig. 1. Equipment for measuring the amplitude of vibration of the round window for a given sound pressure on the eardrum. The sound produced by the movements of the round window is led to the ear of an observer. An earphone is connected to the same tube. If the volume displacement of the round window has the same amplitude, but opposite phase, as the volume displacement of the diaphragm of the earphone, no tone is heard by the observer. In this way it is possible to determine the volume displacement of the round window from the calibration curve of the earphone.

Eustachian tube and a small tube is inserted into the middle ear, just opposite the round window, and cemented into the niche of the round window with an airtight seal. Then the tube and the temporal bone are cemented to a block of lead. A second tube, leading from a loudspeaker is inserted into the ear canal. The sound pressure produced in the ear canal is kept constant by a small condenser microphone. The sound pressure on the eardrum sets the eardrum into vibration, and with it the stapes footplate and the round window. The vibrations of the round window produce volume displacements of the air in the sealed tube. These air displacements are transmitted through a hole in the lead base to the ear of an observer. The intensity of the sound heard by the observer is a measure of the sound transmitted through the whole ear. For the precise measurement of this intensity an earphone is connected to the observation tube. The phase of the current in this earphone may be so adjusted that the air displacements produced by the earphone are exactly opposite to those produced by the movements of the round window. Then if the magnitudes of these two air displacements are the same, all sound in the tube leading to the ear of the observer is cancelled out and the tone is no longer heard. The amplitude and phase of the current in the earphone, after calibration, give a direct measure of the volume displacement of the round window. The difference between this value and those obtained for normal ears[1] is a measure of the conductive hearing loss in the defective ear.

CORRELATION BETWEEN CONDUCTIVE LOSS AND MOBILITY OF THE STAPES UNDER STATIC PRESSURE.

During an operation it is customary to test the mobility of the stapes by using a fine forceps. The forces acting on the stapes in this case are quite different from those produced by a sound.

For a frequency of 1000 cps with a sound pressure on the eardrum of 10^3 dynes/cm^2, *i.e.*, 1 gm/cm^2, the vibration amplitude of the stapes footplate is only about 2.10^{-5} cm.[2] Since this sound pressure is just below the threshold of feeling, an amplitude of 2.10^{-5} cm is near the maximum that can occur in the audible frequency range without danger of damaging the ear. It is possible that even though the stapes may seem fixated for large movements of the forceps, it may be able to conduct, without any loss, the small vibrations of 2.10^{-6} cm.

In order to measure the mobility of the ossicles at autopsy an opening was made in the roof of the tympanic cavity without cutting any ligaments. The opening was large enough to expose the head of the hammer and part of the arch of the stapes. A rubber tube was fitted, with an airtight seal, into the ear canal and the temporal bones and ossicles were photographed, first under a positive pressure of 40 mm. Hg., and then under an equal negative pressure. When these two pictures were observed in a stereoscope, the head of the hammer and the arch of the stapes appeared to project from the plane of the temporal bone, provided the ear was normal (see Fig. 2). If the stapes was fixated, however, the arch of the stapes remained in the plane of the temporal bone and only the bulge between the stapes and incus seemed to project forward (see Fig. 3). The distance of the apparent projection from the plane of the temporal bone is a measure of its lateral displacement.

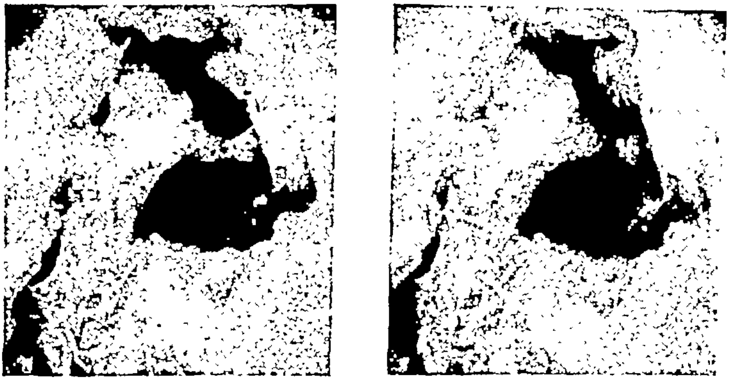

Fig. 2. Stereoscopic photograph of the ossicular chain of the normal ear. Both photographs are of the same temporal bone, first with a pressure of 40 cm. Hg. on the eardrum and then with a suction of 40 cm. Hg. When the pictures are observed in a stereoscope, most of the temporal bone remains stationary and therefore seems to be in a plane. The ossicles are displaced by the changing pressure and they appear, therefore, to jump out from the plane and come closer to the observer.

Since only the horizontal displacements contribute to the three-dimensional stereoscopic effect, for a maximum effect the temporal bone should be turned so that the longitudinal axis of the stapes footplate is vertical and the arch of the stapes, therefore, moves horizontally. Unfortunately shadows produced by the illumination in the photographs of Fig. 2 and Fig. 3 give the surface of the temporal bone a slightly

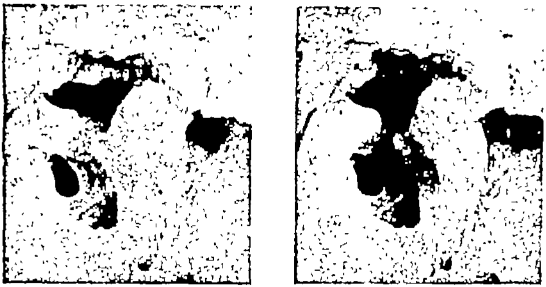

Fig 3. Same as Fig. 2, but for an ear with stapes fixation. The head of the hammer moves about the same degree as in the normal ear, but movement of the stapes is less; therefore, the stapes does not seem to approach the observer to the same extent as in the normal ear.

three-dimensional appearance. In later pictures this effect was overcome by placing a white paper, or mirror, opposite the preparation and taking the picture through a hole in the paper (or mirror).

I had the opportunity to examine four cases of otosclerosis. Comparison of the conductive loss at 1000 cps with the mobility of the stapes, measured by a static pressure, made it clear *that a decrease in the mobility of the stapes was always* accompanied by a hearing loss for the tone; therefore, it seems possible to predict conductive loss for tones if, under static pressure, there is an observable stapes fixation. When the mobility is tested with forceps, however, no quantitative conclusions can be drawn, because the fixation observed with forceps is apparently the same, whether the hearing loss produced by the stapes fixation is 15 or 40 db.

A POSSIBLE WAY TO DETERMINE THE MOBILITY OF THE STAPES IN A PATIENT.

The stereophotographs with suction and pressure in the ear canal proved so useful with anatomical preparations that we attempted to get X-ray photographs of the ossicles of patients, under positive and negative pressures; but this technique was practically useless because the temporal bones differ so greatly from individual to individual that it is difficult to get pictures that show the ossicles. One of the reasons for this failure is that the correct positions for such X-ray pictures have not been worked out.

A better method seemed to be to insert a small endotoscope through a perforation in the eardrum, and to observe the stapes directly. A perforation of about 1 to 2 mm. diameter in the eardrum of a healthy person will heal in a few days. During the war it was customary in some countries to perforate the eardrums of fighter pilots so that sudden altitude changes would cause no discomfort. In the case of patients, however, opinion is divided on the matter of the healing of such perforations; but since a perforation in the eardrum does not affect the threshold of hearing in the middle frequency range,[3] this method seems to be justified in cases of combined hearing loss when a correct diagnosis is very important.

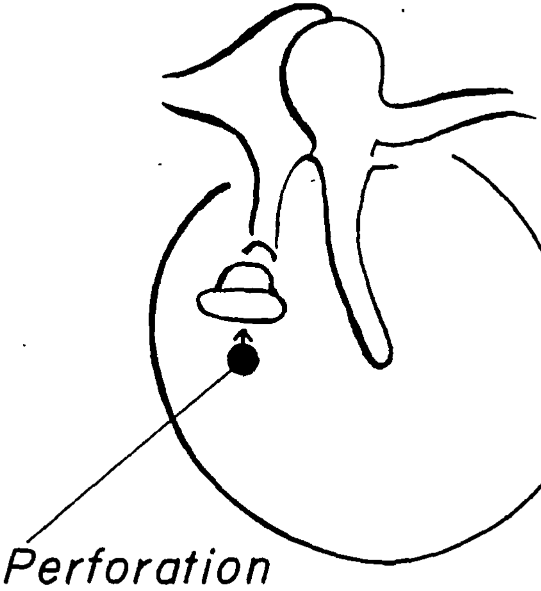

Fig. 4. In order to observe the movement of the stapes in a patient, it is suggested that the eardrum be perforated at the place indicated. The stapes-incus joint can then be observed from below by means of a small endotoscope inserted through the perforation.

A discussion with Dr. Julius Lempert in New York brought out the fact that an endoscope for examination of the middle ear (endotoscope) would probably be of some value. Furthermore, our animal experiments had made it clear that a very small endotoscope would aid in the study of the middle ears of guinea pigs and rats. Consequently we attempted the construction of such an instrument.

Experiments on cadavers showed that the stapes-incus joint can be observed by means of an endotoscope inserted through a hole in the eardrum at the place shown in Fig. 4. The joint is seen from below, as sketched in Fig. 5. On the stapes-incus joint there are two parallel bulges, from which the light is reflected in two sharp streaks. If there is no stapes fixation, pressure acting on the eardrum causes both of these highlights to move, the distance between them remaining the same. The movements are vertical to the longitudinal axis of the stapes footplate. If, however, the stapes is fixated, the distance between the two highlights changes. Thus a stapes fixation can easily be detected.

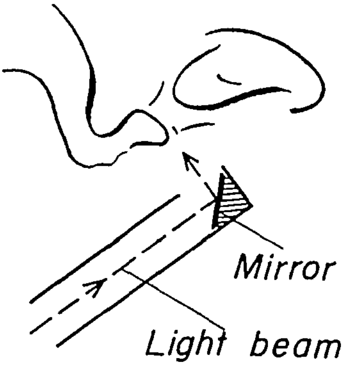

Fig. 5. Path of the light beam in the endotoscope for the observation of the mobility of the stapes.

In order to produce the desired pressure on the eardrum, a test hair of v. Frey was used. The hair was a nylon thread (fish-line leader). It was attached to a small handle, and its other end was blunted with a small ball (see Fig. 6). If this thread is pressed against the eardrum, the pressure on the eardrum increases until the thread begins to bend. Further displacement of the handle does not affect the pressure against the eardrum. By pressing against the eardrum intermittently with this hair, one can observe the mobility of the ossicles for a given pressure. The maximum pressure produced by the test hair can be measured in grams on a balance.

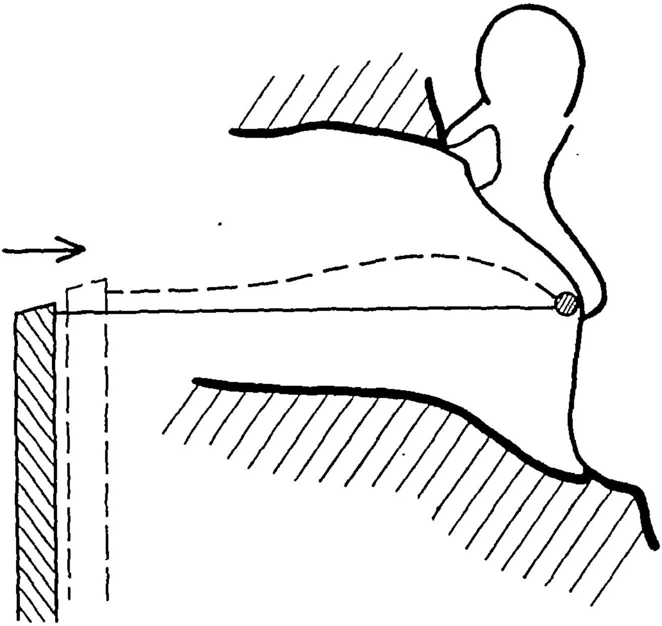

Fig. 6. Test hair for the production of normal pressure on the eardrum, taking advantage of the fact that the pressure produced by the test hair is not dependent upon the deformation of the hair.

It was possible to observe the movement of the stapes on many cadavers without difficulty, except when the ear canal was extremely narrow.

CONSTRUCTION OF AN ENDOTOSCOPE FOR SMALL HOLES.

The main problem in an endoscope is the illumination. If the tube of the endoscope contains a lens, it is generally impossible for the source of illumination to be in the same tube as that through which the observation is made, because light reflected on the surface of the lens is mixed with light from the surface being observed, and the contrast is decreased. We tried to decrease the reflections from the surface of the lens by coating it, but the results were not satisfactory. For this reason, most endoscopes use two tubes, one for observation, the other for illumination. This doubles the thickness of the endoscope.

Since what we wanted to avoid under any circumstances was unnecessary thickness, we attempted the difficult task of building a satisfactory endotoscope in which the same tube is used for observation and illumination, with no disturbing

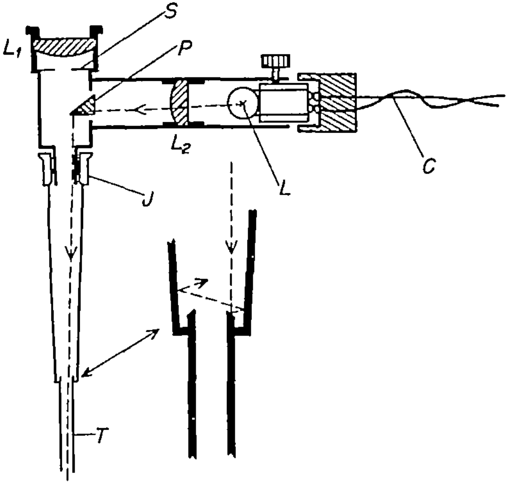

Fig. 7. Schematic drawing of an endotoscope for observation of the middle ear. The tube (T) can be reduced to an outside diameter of 1 to 2 mm., and these tubes can be easily interchanged.

reflections. A schematic drawing of the endotoscope developed is shown in Fig. 7, and a photograph of it is shown in Fig. 8. It was possible to reduce the outside diameter of the tube to 1.5 mm. This tube is made like a hypodermic needle, and can be attached to the T-shaped endotoscope simply by pushing it on, making sterilization quite simple. Having a number of slightly different, easily interchangeable tubes makes the endotoscope adaptable to many purposes. The focus of lens (L_1) was so adjusted that the object at the lower end of the tube appeared sharp. The light from a small bulb (L) was collected by lens (L_2) and directed into the tube by the left-hand edge of a small prism (P). The focus of lens (L_2) and the angle of the prism were so adjusted that a sharp image of the lamp filament was produced on the lower end of the tube (T), illuminating the whole field evenly. For a good observation of the object through such a small hole it seemed important to surround the field of view with a ring of com-

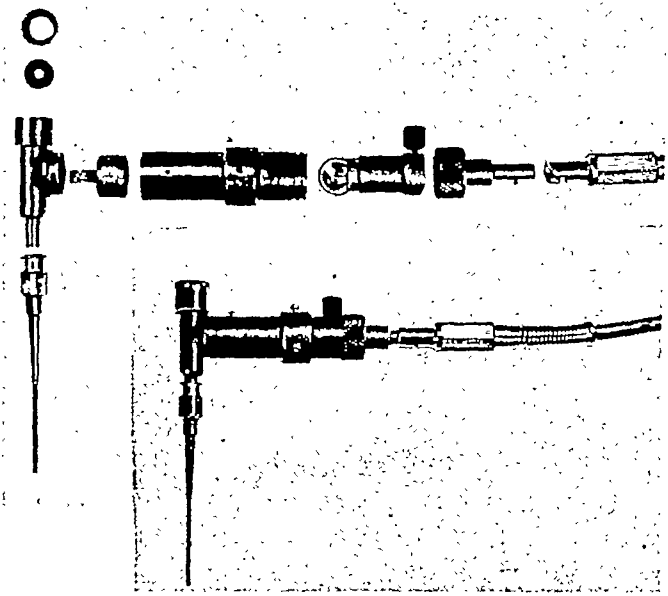

Fig. 8. Photographs of the separate parts of the endotoscope and of the instrument as finally assembled.

plete blackness. For this purpose, the upper end of tube (T) was beveled, as is shown by the enlarged details in Fig. 7. A light beam coming from the lamp is reflected on this beveled edge and absorbed on the side wall of the upper tube, which was covered with fine grooves. A special preparation, Ebonol C,[4] was used to obtain a completely black surface inside the tube, though the coat was less than 1/100 mm. thick. A special stop (S) was provided to eliminate scattered light in the upper parts of the tube.

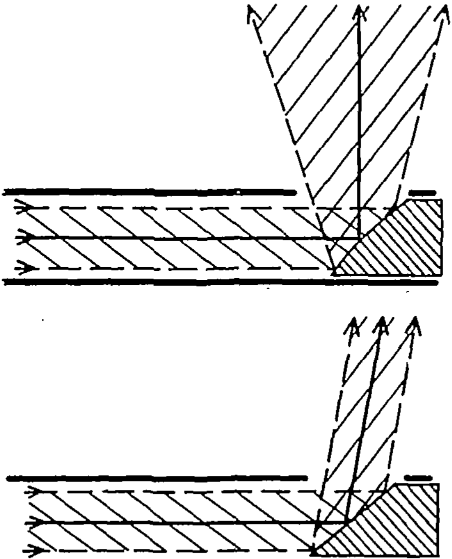

Fig. 8. In order to see around corners, different types of mirrors can be attached to the end of the tube of the endotoscope. The convex mirrors proved especially suitable.

Although at the beginning it seemed impossible to see much by looking through a tube with a diameter of 1 to 2 mm., the experiments demonstrated that after several hours' training it is possible to make out considerable detail.

Four cases of stapes fixation were measured. The decrease in mobility of the stapes for tones of 1000 cps was correlated with a decrease in the mobility of the stapes for static deformations.

For accurate diagnoses it seems possible to introduce into the middle ear a small endotoscope through a perforation of 1 to 2 mm. diameter in the eardrum. With this special endotoscope the mobility of the stapes could be observed, as has been demonstrated on anatomical preparations. It is supposed that this method will prove valuable in the diagnosis of the causes of hearing loss. The small dimensions of the endotoscope make it possible to observe parts of the ear canal that are usually inaccessible. For the examination of the ear canal of experimental animals (guinea pigs, rats, etc.) this endotoscope proved to be indispensable.

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PROCAINE PENICILLIN SOLUTIONS IN THE TREATMENT OF NASAL POLYPOSIS.

SIMON L. RUSKIN, M.D.,
New York, N. Y.

Nasal polyposis has long been one of the most troublesome of the rhinologists' problems. In years past, the complete exenteration of the ethmoid labyrinth was the solution suggested by Halle, and Hajek proposed almost as radical an approach. The recognition of the part played by allergic reactions modified to a great extent the haste for radical surgery. Today it is realized that allergic sensitivity combined with chronic infection is the underlying factor in the causation of nasal polyposis.

Oskar Hisch should be given the credit for first pointing out that the antra were a frequent source for nasal polypi. The histopathology of nasal polypi show primarily two kinds: one, the soft polyp representing the thin sinus mucosa enormously distended by the accumulation of transudate. It is almost a single celled layer of epithelium holding back a volume of transudate, which it cannot absorb. The gradually increasing collection of transudate makes for perpetual growth of the polyp. The rate of absorption cannot keep pace with transudation. There are almost no glands in the sinus mucosa and only very slight secretion. The second type of polyp is the hard polyp formed from tissues of the lateral nasal wall, rich in glands and frequently cystic. This is not the type under consideration in this paper.

Removal of the soft polyp by snare has usually been followed by recurrence, while removal of the hard polyp along with the turbinate tissue from which it springs has less frequently been associated with recurrence.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 15, 1949.

To understand the mechanism and treatment of sinus polyposis, it is well to study not only the histologic appearance but also the reaction of the sinus membrane in the experimental animal. Fabricant has emphasized this in a short, excellent article on animal experimentation and nasal medication. He reported on the "studies which have been made of the absorptive ability of the mucous membrane of the dog's frontal sinuses by the introduction of drugs and other substances. Histamine, nicotine, ephedrine and epinephrine, drugs which institute rapid and pronounced alterations in blood pressure when injected intravenously, were found to be absorbed very slowly from the sinus mucous membrane. The absorption in fact was so slow that no definite changes in blood pressure were detected. A further experiment proved that the absorption from an inflamed mucous membrane is considerably more rapid than from a normal mucous membrane."

Fabricant also points out that "animal studies have demonstrated that various solutions of sodium and calcium penicillin (containing from 1,000 to 5,000 units per cubic centimeter) when applied topically to the respiratory nasal mucous membranes of rabbits every day for 30 to 90 days, have an apparently innocuous effect on nasal tissues. From these animal investigations and from numerous clinical reports on record it appears evident that penicillin solutions administered topically have no deleterious effect on respiratory nasal mucous membranes."

This experimental data is important in our approach to the treatment of nasal polyposis. It is obvious that the location to which treatment of polyposis must be directed is the sinus mucosa. If the transudation of fluid into the sinus subepithelial stroma can be controlled, the growth of the polyp would be stopped and resorption of the transudate slowly accomplished. To do this, a double action must be achieved; that is, the allergic reactivity must be reduced and the infection controlled. In previous years we combined vasoconstrictors with antiseptics, which were used with only very limited benefit. The vasodilatation that followed the vasoconstriction counteracted the benefit of the whole therapy.

Procaine penicillin marked a turning point in nasal therapy. Here we have a preparation that has two active agents that uniquely enhance each other's action in a remarkable fashion. The procaine forms with the penicillin a salt procaine penicillin which is stable in solutions containing 1,000 to 5,000 units per cc. so that solutions can be used at room temperature without undergoing rapid hydrolysis and deterioration. These solutions may also be kept after preparation for prolonged periods. When sodium or potassium penicillin solutions are made hydrolysis sets in directly, since the sodium enters into a reversible reaction whereby sodium hydroxide is formed, which reacts with the penicillin to cause progressive deterioration. When, however, procaine penicillin is put into solution, the tendency to hydrolyze is negligible. It is this stable characteristic that makes it practically the ideal medication for instillation into the nasal sinuses.

The rate of absorption for the sinuses is so slow that when procaine penicillin solution of 3,000 to 5,000 units per cc. is instilled, it remains there for at least one to three days and, at times, a full week. This can be observed during the refilling when the slightly yellowish solution is displaced by the fresh solution. Frequently, crystals of procaine penicillin can be seen in the return flow a week after the original instillation.

Since procaine is an oil soluble base, its absorption by the mucosa is particularly slow, thus producing a depot action within the sinus. The presence of 3,000 to 5,000 units of penicillin in direct contact with the sinus mucosa for several days to a week produces a perfectly enormous dosage at the infected site. Usually one to two units per cc. of blood is considered adequate systemic dosage. In nasal polyposis, we are dealing with a localized infection that is practically not reached by systemic penicillin therapy at all. Higher dosages than 3,000 to 5,000 units per cc. within the sinus are not needed and would be wasteful. This prolonged effect for procaine penicillin is not obtained with sodium penicillin since, at body temperature, sodium penicillin is hydrolyzed in three to four hours and the reservoir effect is not obtained.

Another factor of unique benefit in the procaine penicillin is the antihistamine action of procaine. This has not received all the attention it deserves. Frommel and his Swiss co-workers showed that procaine protected experimental animals against lethal doses of histamine aerosols and counteracted the effect of histamine on the terminal ileum. Clinically, it can be readily observed that following filling of the antra with procaine penicillin solutions there is decongestion of the mucosa of the lateral nasal wall with improved aeration of the nasal passages and improved drainage from the frontal, ethmoid and sphenoid sinuses. Filling of the sphenoid sinuses is an excellent collateral aid.

The antra are filled through the natural ostia. I use the Ruskin modification of the Yankauer cannula. This has simplified the technique considerably. For the preparation of the procaine penicillin solution, I add 20 cc. of procaine ascorbate solution to the vial containing 100,000 units of sodium penicillin. This forms the procaine penicillin and sodium ascorbate directly in the vial. The sodium ascorbate is useful in the solution since the ascorbate enhances penicillin action, as also shown by Jalili at Fuad University, and the ascorbate likewise has an antihistamine effect, as I have previously reported. An alternative method of preparation is the further dilution of the commercially available, ready to use, suspension solutions of procaine penicillin supplied by Squibb and Co., and Wyeth. One cubic centimeter of the solution-suspen- Co., and Wyeth. One cc. of the solution-suspension of procaine penicillin, containing 300,000 units, is diluted with two ounces (60 cc) sterile saline. Ten cc. of this solution are instilled into each antrum and 5 cc. into each sphenoid sinus. Thus, one ounce is used for a complete treatment. This gives the patient 150,000 units in direct apposition to the sinus mucosa for a period actually of one to seven days. The use of the dilute solution in 10 cc. volume is preferable to high dosage such as the 300,000 units in 1 cc. because the 10 cc. produces a continuous drip through the natural ostium, bathing the lateral nasal wall, nasopharynx and pharynx. This produces a generalized decongestion of the nasal mucosa. Under this treatment chronic sinus conditions of very long standing have shown striking improvement. It has, in my experience, eliminated sinus surgery to a remarkable degree.

The case here reported has been taken from a series of 60, to typify the results obtained.

Mr. L, age 60, had been suffering for over 20 years from recurrent sinus infections and polyposis. He had had three polypectomies and when advised to have a fourth, refused further surgery. At the time of the examination he was complaining of bilateral frontal headache, nasal discharge and nasal obstruction. His difficulties were worse at night when his nasal obstruction disturbed his sleep. He frequently was aroused several times during the night by the nasal obstruction. He had been using various nasal vasoconstrictors at night for years.

Nasal examination revealed multiple polypi in both middle meati, obstructing also both nasal fossae.

Following anesthetization of both sphenopalatine ganglia, each antrum was filled with 10 cc. of procaine penicillin solution containing 5,000 units per cc. prepared as above. This was repeated daily for three days, then every other day for six days and, after that, once weekly for two instillations. The patient also received simultaneous injections of calcium ascorbate (Calscorbate) intramuscularly. After the second day the patient reported that he was able to sleep through the night. The following refilling showed procaine penicillin crystals in the return flow. He felt very much more comfortable and, after one week, the polypi had shrunk to less than half their original size. After the third week the nose was clear and free from polypi. The patient was discharged and has remained free from nasal complaint or polyp recurrence for six months.

SUMMARY.

1. Nasal polyposis is amenable to conservative treatment by the use of solutions of procaine penicillin, 3,000 to 5,000 units per cc., when instilled into the nasal sinuses.

2. Procaine penicillin solutions provide a prolonged acting penicillin effect combined with the antihistaminic effect of procaine in decongesting the nasal mucosa. It is a great advance in nasal therapy.

32 East 67th Street.

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DISLOCATION OF THE LARYNX.*†

LENNART GISSELSSON, M.D.,
Lund, Sweden.

Judging by the literature, dislocations of the larynx are very rare indeed. The joints between the cartilages of the larynx are rigid and well protected so that only a heavy blow or rather extreme compression will cause a dislocation of these joints. Laryngeal dislocations are as a rule caused by a direct blow against the larynx, when the resultant dislocation will most frequently be accompanied by a fracture of one of the cartilages. The joint most liable to dislocate is naturally the *most flexible one, the cricoarytenoid. Only one case of* dislocation of the cricothyroid articulation could be traced in the literature. Jackson and Jackson describe a man who, in an attempt to hang himself, dislocated both arytenoid cartilages and the cricothyroid articulation. The report of a further case, therefore, seems warranted.

CASE REPORT.

In an attempt to suppress a yawn, the patient, a 43-year-old man, had experienced a click in the larynx, and since then swallowing caused an intense pain in the left half of the throat. Examination showed that although the laryngeal prominence was still in the median plane, the *posterior part of* the thyroid cartilage protruded to the left. Laryngoscopy revealed the aryepiglottic fold to be somewhat extended on the right side but shortened on the left side. The voice and the movement of the vocal cords were apparently normal. The patient was X-rayed but no ossification of the laryngeal cartilages was observed, so that it was difficult to satisfactorily explain his trouble. The only observation made was that the airway in the larynx was displaced to the left (see Fig. 1); however, on more careful palpation the inferior cornu of the thyroid cartilage could be felt on the left side, and it was found that this was

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*Read at the meeting of the Swedish Otolaryngological Association, Stockholm, Sweden, November, 1947.

†From Department for Diseases of Nose, Throat and Ear of the University of Lund (Department Head: Prof. Gösta Dohlman).

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 6, 1949.

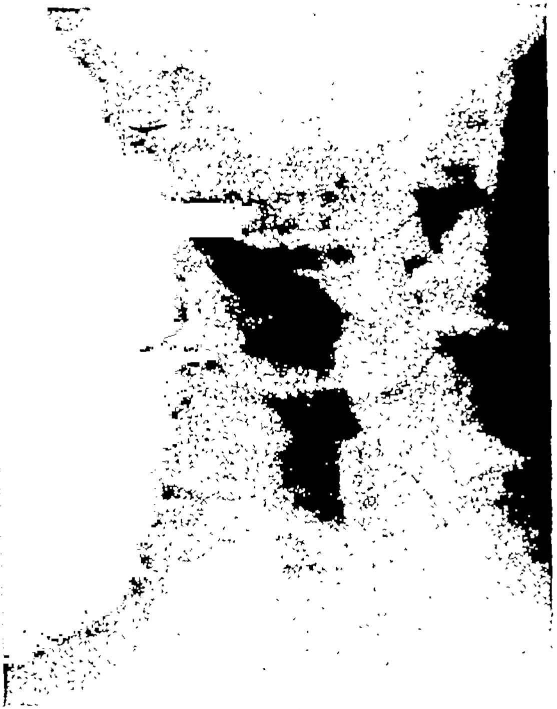

Fig. 1.

where the patient felt the pain. On the right side the inferior cornu could not be palpated; furthermore, the caudal margin of the thyroid cartilage with its inferior tubercle could be palpated on the left side but not on the right. The case was, therefore, diagnosed as a cricothyroidal dislocation. Local anesthesia was induced and pressure applied to the left side of the thyroid cartilage without any result, but when the right side of the thyroid cartilage was then lifted cranially and gentle pressure applied to the left side, a click was heard. The thyroid cartilage had snapped into normal position, and the patient was immediately able to swallow without discomfort. He was X-rayed again after this reposition, and the X-ray plate showed that the airway now passed straight through the larynx (see Fig. 2).

It must, therefore, have been a question of dislocation of the cricothyroid articulation and of the nature diagrammatically illustrated in the right sketch of Fig. 3. Why is this dislocation so rare? It is probably because in most individuals the joint between the thyroid and cricoid cartilages resembles more a symphysis than a joint in the general con-

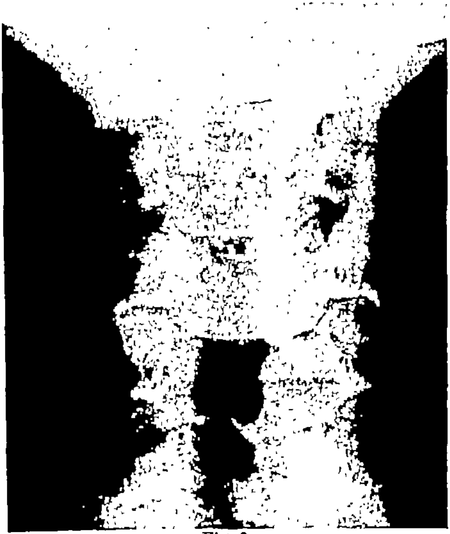

Fig. 2.

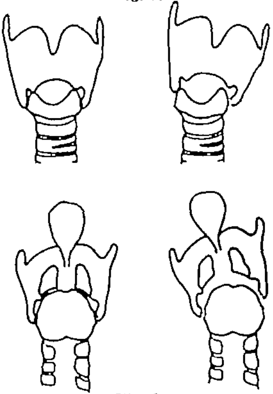

Fig. 3.

ception of the word. Cases with a fully developed joint have been described, even with a cartilaginous plate in the form of a disk between articulating surfaces. In the present case of laryngeal dislocation it must be a question of a proper joint, and the usually very tight ligaments in the capsule must be very elastic. Another reason why laryngeal dislocations are so rare is perhaps the unawareness of their occurrence. It is possible that some of the distinct pains in the throat which cannot be satisfactorily explained and are often diagnosed as neuresthenia, are some form of distortion or dysfunction in the cricothyroid joint.

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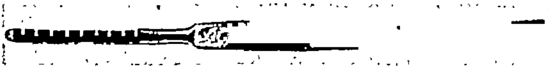
A NEW BLUNT END STRAIGHT SAW-KNIFE FOR LOWERING A CARTILAGINOUS NASAL HUMP.*

M. William Mootnick, M.D.,
New York, N. Y.

During my years of practice in the field of rhinoplasty, I occasionally encountered some technical difficulty in the correction involving a cartilaginous nasal hump and where the bony nasal bridge does not require any surgery, I found it very difficult or impossible to take a positive grip with an ordinary blunt end straight knife on the perichondrium covering the upper lateral cartilages, including the dorsal portion of the nasal septum.

To overcome this difficulty I decided to put teeth into the same instrument, enabling the latter to take a firm, positive bite into the perichondrium covered cartilaginous hump with ease. The operative procedure may be completed with the saw knife or followed with the blunt end straight knife.



This instrument could also be used to advantage in lowering the cartilaginous portion of a normal profile after shortening the nasal tip in a long nose to make room for the excess nasal skin which thickens through contraction after operation.

11 West 42nd Street.

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*Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 4, 1940.

KENFIELD MEMORIAL SCHOLARSHIP.

In 1937, a sum of money was subscribed in memory of Miss Coralie N. Kenfield, of San Francisco, Calif., a teacher well known throughout the United States for her high ideals and advanced methods in teaching lip reading. This money, placed in the Kenfield Memorial Fund, is administered by the American Hearing Society and provides for an annual scholarship. The amount of the Kenfield Memorial Scholarship for 1950 is $100.00.

Applications for the scholarship will be considered from any resident of the United States who desires to teach lip reading (speech reading), with or without other types of hearing, and speech therapy to the hard of hearing, and who can meet the following requirements:

Personal: Well adjusted individual with a pleasing personality, legible lips, a good speech pattern and no unpleasant mannerisms.

Education: College graduate with a major in education, psychology, and/or speech, plus professional training in lip reading; 30 clock hours of private instruction under an approved teacher of lip reading or 60 clock hours of instruction in public school classes under an approved teacher of lip reading.

The winner of the scholarship may take the teacher training course from any normal training teacher or school or university in the United States offering a course acceptable to the Teachers' Committee of the American Hearing Society. The scholarship must be used within one year from the date the award is made.

Applicants must be prospective teachers of lip reading to the hard of hearing. Those already teaching lip reading cannot be considered.

Applications must be filed between March 1 and May 1, 1950, with: Miss Rose V. Feilbach, Chairman, Teachers' Committee, 1157 North Columbus Street, Arlington, Va.

BOOK REVIEWS.

Public School Audiometry: Principles and Methods. By Loraine Anson Dahl, formerly Field Supervisor, Wisconsin State-Wide Hearing Survey; Supervisor, Hearng Test Service, Speech Clinic, Purdue University; Research Associate, Department of Otolaryngology, University of Iowa. With Foreword by Wendell Johnson, Ph.D., Director of Speech, University of Illinois. Two hundred ninety pages with Index and Bibliography. Danville, Ill.: The Interstate Printers and Publishers, 1949.

This book is made up of three principal parts: *1.* Factors to Be Considered in a Hearing Conservation Program, 2. Creating Active Interest in the Conservation of Hearing, and *3.* Techniques and Principles of Audiometry. It also includes a Manual of Instruction of Audiometer Procedures in Public Audiometry, plus three sets of references.

The book should be helpful to those engaged in hearing conservation programs and to those who are on the periphery of such problems, such as health rehabilitation and guidance workers. The author is to be commended for emphasizing the follow-up in group testing which is too often neglected in public conservation programs. The suggestions for creating interest in a program of hearing conservation should be helpful to those who are inaugurating such a program. The forms at the end of the book should be very useful because they anticipate a great deal of detail involved in the program; however, the book leaves much to be desired, not so much because of what it says, but because of many errors of omission.

A perusal of the three bibliographies suggests that the author has failed to take adequate notice of recent developments in the field. This is surprising, since the book is dated June, 1949. Except for one or two items, the bibliography goes back to dates previous to 1944. Even if the author did not intend to cover some of the recent developments in detail, it would have been helpful to her readers to be guided to recent source material. A few illustrations of omission will serve to emphasize this point. In discussing classifications of audiograms, no mention is made of Carhart's useful method described in 1945. There is no reference to the Massachusetts Hearing Tests. Recent books which antedate the publication of this book, such as Brentano's book and the volume edited by Hallowell Davis, "Hearing and Deafness" are not mentioned. Under "periodicals" the author fails to include the *American Annals of the Deaf, Hearing News* and the *Journal of Exceptional Children.* In a survey of the "etiology" and "causes" of deafness, which ranges from congenital deafness in children to presbycusis in adults, it is surprising that no mention is made of otosclerosis or the fenestration operation. It would have been extremely valuable for the reader to have his attention called to recent developments in hearing aids and articulation tests, particularly as part of the follow-up program.

In the section on techniques and principles of audiometry nothing is said about the diagnostic value of bone conduction tests as part of the follow-up. In discussing the technique of testing in cases of unilateral impairment the author fails to point out the technique of masking.

There are also errors of proof-reading. For example, on page 287, which is the first page of the index, we have the reference "Davis, H." and on page 283 the same author is listed under the H's as "Hallowell, D." It is also amazing that the book has been put out without a table of contents. This reviewer could continue this list of omissions and inadequate treatment, both in the text and in the bibliography, but the examples given above indicate that the book is ready for some revision.

S. R. S.

Your Nasal Sinuses and Their Disorders. By Albert P. Seltzer, M.D., M.Sc., Sc.D.(Med.), F.A.C.S., F.I.C.S. Assistant Professor in Otolaryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Otolaryngologist, Mt. Sinai Hospital, Philadelphia; Associate Chief in Ear, Nose and Throat, St. Luke's Medical Center; Chief of Staff in Plastic and Maxillo-Facial Surgery at Douglass-Mercy Hospital, Philadelphia; Chief of Staff of Plastic and Reconstructive Surgery, Community Hospital, Philadelphia. One hundred fifty-five pages with Index. New York, N. Y.: Froben Press, 1949. Price $2.50.

This book is presumably written for the laity. It carries one piece of advice, viz., "If you think you have sinusitis consult a nose and throat specialist," and two admonitions: *1.* "Don't diagnose your own case." 2. "Don't treat yourself."

The author has used 152 pages and 24 illustrations to make these three points. One wonders what is accomplished by any discussion on the evolution and prenatal development of the nose and sinuses in such a book—for the layman.

One questions the wisdom of a discussion in a textbook of this kind, of emotional factors in sinus disease, or of the symptomatology of sinusitis, as is done in the chapter entitled, "What Is Your Complaint?" Surely this is urging the layman to make his own diagnosis, and the patient, with the little knowledge gleaned from this book becomes a nuisance to the otolaryngologist whom he finally consults.

The 24 illustrations are mostly without point, are undignified and have nothing to recommend them.

All in all, it seems this book will do more harm than good.

T. E. W.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of January 1, 1950.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver) ; Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone; Radioear Permo-Magnetic Multipower.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc.. 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Ravox—*Jour. A. M. A.*, 113:18 (Oct. 28), 1939.

Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
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Council Meeting, Hotel Statler, New York City, Jan. 6, 1950.
Combined Meeting, Southern and Middle Sections, Peabody Hotel, Memphis, Tenn., Jan. 16-17, 1950.
Western Section, Medical Society Bulding, Los Angeles, Calif., Jan. 21-22, 1950.

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Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, Pa.

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AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

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THE

LARYNGOSCOPE.

VOL. LX FEBRUARY, 1950. No. 2

ANTIBIOTICS AND SINUS INFECTIONS.*†

F. W. DAVISON, M.D.,
Danville, Pa.

A comprehensive discussion of the treatment of sinus infections would include consideration of such factors as abnormal anatomy, drainage, immunity, allergy, metabolic and endocrine deficiencies, nutrition and the use of the antibiotics. I shall limit my remarks to the latter subject.

During the past two years clinical observation has made me increasingly aware of the fact that I was not seeing as satisfactory response to the use of penicillin as I had observed two to three years ago, even though rather large doses have been used during 1949. In an effort to substantiate and explain this clinical impression, I have reviewed the charts of patients with sinus infection who have had cultures from pus in the nose made on penicillin sensitivity plates during the past five years. The information obtained was rather startling, as is shown in Table 1.

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., New York City, N. Y., Jan. 5, 1950.

†From the Department of Otolaryngology and Broncho-Esophagology, the Geisinger Memorial Hospital Danville, Pa.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

TABLE 1.

CULTURES FROM PUS IN THE NOSE IN CASES OF ACUTE OR CHRONIC SUPPURATIVE SINUSITIS.

BLOOD AGAR PLATES TREATED WITH PENICILLIN.

Year	Number Cultures	Gram Positive Cocci Only
1945	10	80% sensitive to 0.2 unit per cc. (inhibited by)
1946	44	82% sensitive to 0.2 unit per cc.
1947	33	60% sensitive to 0.2 unit per cc.
1948	46	26% sensitive to 0.2 unit per cc.
1949	48	12% sensitive to 0.2 unit per cc.

My interpretation of the data in Table 1 is as follows: Because of the widespread use of penicillin dust in "sniffers," penicillin nose drops, penicillin aerosol inhalations and *inadequate dosage* by mouth or by injection, the more sensitive organisms have been killed, and the more resistant strains have survived to be coughed and sneezed onto friends and relatives. Thus the bacterial flora of the upper respiratory tract has in a period of three short years become relatively resistant to the action of penicillin.

Dr. Fleming[1] said in 1946, "It is to be hoped that penicillin will not be abused as were the sulfonamides. . . . It is the administration of too small doses which leads to the production of resistant strains of bacteria, so the rule in penicillin treatment should be to give enough."

Pus for these cultures was obtained from the interior of the nose by means of the suction collector shown in Fig. 1. The organisms demonstrated were various types of staphylococci; streptococci and pneumococci, grown aerobically on blood agar plates treated with various concentrations of penicillin, with one untreated control plate. I am not stating that these cultures represent the infecting organism, the pathogen solely responsible for the disease, but they do represent the bacterial flora found in pus in the noses of patients having obvious suppurative sinus infection, and show the decreasing sensitivity of these organisms to the action of penicillin.

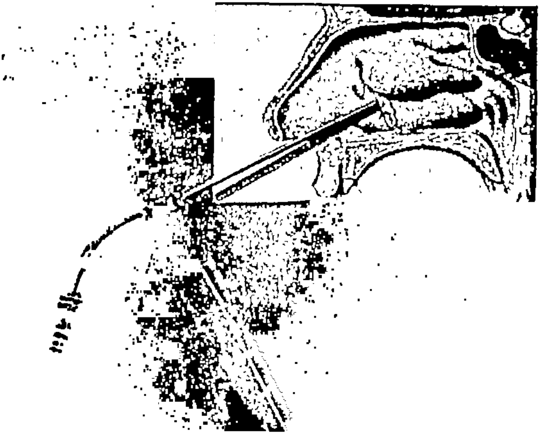

Fig. 1. A suction-collector obtaining an uncontaminated specimen of pus from the nose for smears and cultures.

This study has at least one serious flaw: no anaerobic cultures were made. The preparation of this paper made me find some articles that I had previously overlooked. Those written by Galloway,[2] Williams and Heilman,[3] Pastore and Williams[4] impressed upon me the importance of the microaerophilic streptococci as causal agents of sinus infections and their complications.

My belief that the gram positive cocci demonstrated by culture are at least some of the pathogens is strengthened by the smears which show many cocci mixed with the neutrophiles. Quite often two organisms, usually a staphylococcus and a streptococcus, were found in a given sample of pus, and their behavior with respect to penicillin sensitivity plates varied considerably, as is shown by Table 2.

TABLE 2.
CULTURE ON PENICILLIN TREATED BLOOD AGAR PLATES.

	0.2	0.5	1.0 Units Per Cc.
Staph. aureus	4+	4+	4+
Strep. viridans	1+	0	0

+ equals growth. 0 equals no growth.

This method of testing bacterial sensitivity has also been used by Nichols and Needham.[5]

In 1947, I wrote a paper,[6] based on cases treated during 1946, indicating that nearly all patients with acute, and some patients with chronic suppurative sinusitis could be cured by intramuscular injection of penicillin, using 50,000 units every three hours, day and night, for a period of seven days, *providing the infection was due to organisms sensitive to penicillin in concentration of 0.2 unit per cc.* This is perhaps still true, but only 12 per cent of the organisms cultured from pus in the nose are at present writing sensitive to this concentration (at least in Central Pennsylvania).

The frequency of penicillin-resistant infections is often not appreciated, and I have seen the diagnosis of "Virus X" infection used more than once to explain the cause of a penicillin-resistant infection. Barber[7] and her co-worker in England reported an incidence of 14.1 per cent resistant strains of staph. pyogenes in 1946 with strains resistant to 10 units, increasing to 38 per cent in 1947, and to 59 per cent in 1948. Nichols and Needham,[5] in 1949, reported that 68 per cent of 50 strains of staph. aureus were resistant to 1.6 units of penicillin.

TABLE 3.
1949 PENCILLIN SENSITIVITY CULTURES FROM PUS IN THE NOSE.

Number Cultures	Concentration of Penicillin in Units Per Cc.	Per Cent of Organisms Inhibited
48	0.2	12%
65	0.5	43%
65	1.0	55%
21	3.0	81%

The data in Table 3 suggest that blood levels in 1949 have to be 15 times as high as in 1946 in order to inhibit the growth of 81 per cent of the gram positive cocci found in pus in the nose.

These data cast doubt on the current idea that an intramuscular injection of three or four hundred thousand units every 24 hours will be effective as treatment for most respiratory infections, and make ludicrous the reference to 0.03 unit as the "minimal effective therapeutic level."

Even the pneumococcus has become more resistant, so that in 1949 it frequently grows on plates containing penicillin 0.5 unit per cc., as shown in Table 4.

TABLE 4.

SENSITIVITY CULTURES FROM PUS IN THE NOSE.

	Penicillin			Dihydro-Streptomycin			Aureomycin		
	0.2	0.5	1.0	5	10	25	1	2	5
Staph. aureus	2+	2+	2+	1+	1+	1+	1+	0	0
Dip. pneumoniae	3+	1+	0	0	0	0	0	0	0

+ equals growth. 0 equals no growth.

The following quotation from Sir Alexander Fleming's book[4] entitled, "Penicillin, Its Practical Application," should serve to keep our thinking straight: "There are a few simple rules for the use of penicillin in treatment of bacterial infections. These may seem too simple — even childish — but experience in watching penicillin being used has made it clear that these rules are often broken with resulting disappointment.

"*1.* It should be used only when there is an infection by a penicillin-sensitive microbe.

"*2.* Penicillin must be administered in such a way that it comes in contact with the infecting microbe.

"*3.* The dose should be such that in the infected area the concentration of penicillin is sufficient to destroy the bacteria.

"*4.* The treatment should be persisted in until the infection is defeated."

Regardless of the numerous articles advocating the topical use of penicillin as nose drops, aerosol inhalations, by injection into antra and by displacement, the words of Dr. Fleming have led me to ignore these methods because they do not live up to his four cardinal principles. Since penicillin must be administered in such a way that it comes in contact with the infecting microbe, and because the germs are located at various depths *within* the tissues where they can be reached only by blood-borne penicillin, I use *only intramuscular injections* in the treatment of sinus infections.

It is not enough to say "penicillin-sensitive." For intelligent treatment we must know *how* sensitive the organism is if we are going to be able to determine when to use penicillin and *how much to use.* The following quotation from Marvin[9] indicates how misleading hasty conclusions may be: "Loewe deserves great credit for having demonstrated the value of

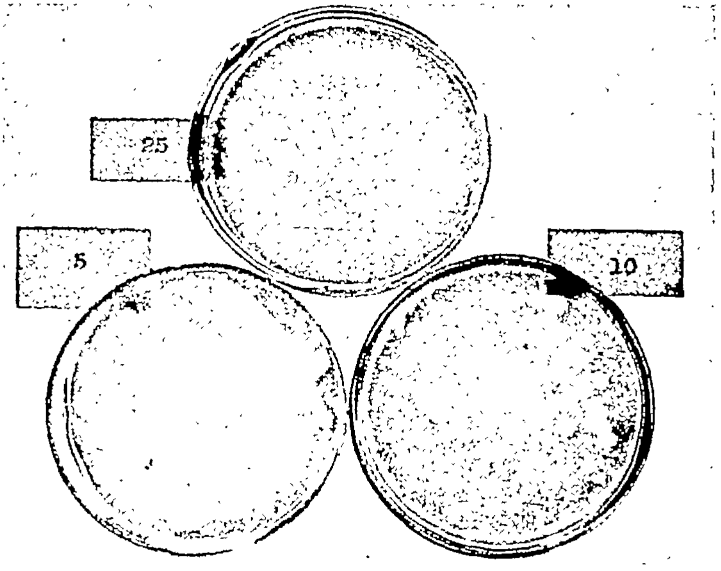

Fig. 2. The growth of a hemolytic streptococcus on blood agar plates containing 5, 10 and 25 micrograms of dihydro-streptomycin per cc. Note complete inhibition of growth on the 25 microgram plate.

large doses of penicillin for subacute bacterial endocarditis after the Penicillin Committee had officially declared it to be of no value." In some cases of sinus infection I use 300,000 units of procaine penicillin with 100,000 units of soluble penicillin every three hours, when smaller doses fail to clear up the infection, and I prefer the aqueous suspension. I think this will maintain a serum concentration of 3.0 units or more, but I have no exact data.

In choosing a chemotherapeutic agent, *the size of the dose* and the *duration of treatment* are both of importance. The site of the infection as well as the degree of penicillin-sensitivity of the organism will partially determine the duration of treatment as is indicated by Table 5 taken from Dr. Fleming's book,[6] published in 1946.

TABLE 5.

AVERAGE MINIMUM DURATION OF DAYS OF TREATMENT FOR PATIENTS RECEIVING 60,000 UNITS EVERY THREE HOURS.

Uncomplicated gonorrhea	1 day
Vincent's angina	2 days
Erysipelas, impetigo	3 days
Carbuncles, cellulitis	5 days
Breast abscesses	7 days
Otitis media and mastoiditis	8 to 10 days
Sinusitis	10 to 12 days
Complicated septicemias	12 to 21 days
Endocarditis	21 to 28 days

Our therapy should not be dictated by the size of the dose marketed in 1 cc. ampules and alleged to be adequate daily dose for most infections. The 96-hour advertising has also resulted in much undertreatment.

When "Penicillin-resistant" infections are encountered, one naturally thinks of other antibiotics. We started making cultures on streptomycin-treated plates in October, 1947, using 5, 10 and 25 micrograms per cc. Fig. 2 shows the appearance of these plates. As soon as dihydro-streptomycin became available, sensitivity cultures were made with this antibiotic and we found that its antibacterial effect with reference to the cocci was the same as that of streptomycin in the same concentration.

TABLE 6.

1949 DIHYDRO-STREPTOMYCIN SENSITIVITY CULTURES FROM PUS IN THE NOSE.

Number Cultures	Concentration of Dihydro-Streptomycin in Micrograms Per Cc.	Per Cent Inhibited
65	5	66%
65	10	86%
65	25	93%

Table 6 shows that 86 per cent of the cocci are inhibited by 10 micrograms of dihydro-streptomycin per cc. blood agar. Thompsett[10] has shown that serum concentrations of this amount of streptomycin can be produced by giving 10 milligrams per kilogram body weight every 12 hours. This means that an effective dose for a 150-pound person is 0.7 gm. every 12 hours, and according to his data this dose should seldom, if ever, produce toxic reactions; however, Thompsett was reporting only the toxic effects of streptomycin. Recent information obtained from Dr. Glorig[11] indicates that dihydro-streptomycin can have a *delayed* toxic effect on the cochlear division of the VIIIth nerve. Thirteen per cent of his patients given 2 gm. of dihydro-streptomycin daily for at least 40 days had severe hearing loss. I have seen one patient who received only 22.6 gm. of dihydro-streptomycin who developed hearing loss and severe persistent tinnitus two months later; hence, we must be cautious in administration of dihydro-streptomycin and be certain that renal function is not impaired.

This is unfortunate because our cultures and clinical observations indicate that at present dihydro-streptomycin is much more effective than penicillin in usual dose.

The results of our aureomycin sensitivity cultures are shown in Table 7.

TABLE 7.

1949 AUREOMYCIN SENSITIVITY CULTURES MADE FROM PUS IN THE NOSE.

Number Cultures	Concentration of Aureomycin in Micrograms Per Cc.	Per Cent Inhibited
57	1	86%
57	2	95%
57	5	98%

Herrell and Heilman[12] state that a serum concentration between 2 and 4 micrograms can be maintained by giving 750 mg. of aureomycin by mouth every six hours; therefore, I have used this dose and interval when aureomycin appeared to be the drug of choice. It is expensive and may produce nausea, vomiting and diarrhea, so there are some relative *contraindications to its use.*

The majority of our cultures indicate that chloromycetin is *not* so effective as aureomycin against the gram positive cocci. Table 8 is an example.

TABLE 8.

SENSITIVITY CULTURES FROM PUS IN THE NOSE.

	Penicillin			Dihydro-Streptomycin		
	0.2	0.5	1.0	5	10	25
Staph. aureus	4+	4+	4+	0	0	0
Strep. viridans	4+	4+	1+	1+	0	0
	Aureomycin			Chloromycetin		
	1	2	5	1	2	5
Staph. aureus	1+	0	0	4+	3+	3+
Strep. viridans	4+	4+	1+	4+	4+	0

Table 8 also indicates how sensitivity cultures can help one decide which antibiotic to use. Because of its cost and poor antibacterial effect with reference to the gram positive cocci, I have not used chloromycetin.

I no longer use sulfadiazine except for meningitis or H. influenzae infections. The superiority of penicillin over sulfadiazine was shown by Wilcox[13] and in 42 per cent of his cases the use of sulfadiazine was discontinued because of toxic symptoms or failure to cure the infection.

DISCUSSION.

I have no statistical data to present showing results of treatment with penicillin alone, because most patients received dihydro-streptomycin as well as large doses of penicillin. The doses I now use for a 150-pound person are procaine penicillin, G 300,000 U combined with 100,000 U crystalline penicillin every three or every six hours, day and night. I subscribe to Dr. Fleming's statement that maximal antibiotic activity is dependent upon high sustained serum concentrations. In addition, many patients were given dihydro-streptomycin 0.7 gm. every 12 hours and treatment was continued for seven to 10 days. This treatment program has cleared up some long standing chronic infections without operative treatment other than puncture and lavage of pus filled antra. Other patients with more extensive disease required judicious but minimal surgery.

CONCLUSIONS.

Most of the gram positive cocci found in the nose are at present (1949) relatively insensitive to the action of penicillin. Penicillin is still effective in treatment of sinus infections if doses as large as 1.6 to 3.2 million units are given daily for a period of seven to 10 days. Sensitivity cultures on blood agar plates are a useful method of determining which antibiotic to use and the size of the dose to be used.

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THE SURGICAL TREATMENT OF BILATERAL POSTICUS PARALYSIS OF THE LARYNX.*

LOUIS H. CLERF, M.D.,
Philadelphia, Pa.

Prior to King's report on the treatment of bilateral paralysis of the larynx by transposition of the omohyoid muscle to the arytenoid cartilage, the surgical results were generally unsatisfactory. While the immediate effects often were favorable, after fibrosis occurred one was confronted with cicatricial as well as paralytic stenosis.

My first attempt at transposition of the omohyoid to the arytenoid cartilage was made in 1940, employing the technique described by King.[1] It proved to be only partially successful, and I attributed this to failure to shorten the omohyoid muscle sufficiently. Later the procedure was repeated on the opposite side and a satisfactory airway was secured.

In one of the early cases I could not identify the omohyoid muscle which had been incised when the thyroidectomy was done. After mobilizing the arytenoid cartilage it was secured with chromic catgut sutures to the posterior border of the wing of the thyroid cartilage. The result in this case was the best I ever had secured, and following this experience I no longer transposed the omohyoid muscle but secured the mobilized arytenoid cartilage with a silk suture passed through a drilled hole made near the posterior border of the corresponding wing of the thyroid cartilage. Motion pictures of the larynx made before and after operation in a number of these cases revealed conclusively that the arytenoid cartilage and

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., New York City, N. Y., Jan. 5, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

vocal cord on the operated side remained immobile and the only movement noted during phonation occurred on the unoperated side. This observation also had been made by others and it now is generally believed that the success of this plan of surgical treatment is dependent upon free mobilization of the arytenoid and its lateral fixation, and that the rôle of the omohyoid muscle is of little or no importance.

In 1941, Kelly[2] reported his results by arytenoidectomy through a resected window in the ala of the thyroid cartilage and fixation of the cord. McCall and Gardner[3] recommended transillumination of the larynx to aid in locating the position of the arytenoid and, also, the employment of a suture to bring the arytenoid through the resected window. Orton[4] advised removal of the posterior portion of the wing of the thyroid cartilage to facilitate the approach to the arytenoid cartilage. Woodman[5] recommended partial arytenoidectomy with retention of the vocal process and fixation of the vocal cord to the inferior cornua of the thyroid cartilage. Thornell[6] reported an intralaryngeal approach for arytenoidectomy in bilateral abductor paralysis and reported his results in three cases. The most recent report on this subject was presented by Georges Perello,[7] who described a new technique which consists of making a window in the thyroid cartilage and suturing the superior end of the sternothyroid muscle to the internal fasciculus of the thyroarytenoideus muscle or to the true vocal cord.

Since I have had no personal experience with the latter procedures, no further comment shall be made other than to state that the advocates of these have reported satisfactory results. I have employed the King technique with certain modifications in all my cases. In a report[8] in 1947, based on 32 operated cases, four were unsuccessful after the first operation, but in three of these a satisfactory result was secured following operation on the opposite side. The remaining case, a teacher, refused a second operation, as she preferred to use a valved tracheal cannula and retain a good speaking voice. In these four, the original King technique in which the omohyoid muscle was transposed was used. I am in accord with Dr. King that this method is not satisfactory.[9]

Technique: In patients wearing a tracheal cannula one may insert an intratracheal tube preoperatively through the tracheotomy fistula, thus keeping the anesthetist out of the field of operation and diminishing the dangers of wound contamination. If no tracheotomy has been performed, one either can insert an intratracheal airway through the larynx preoperatively and perform tracheotomy at the termination of the operation or the tracheotomy may be performed preoperatively and an airway inserted through the tracheal stoma. While placement of an airway in the larynx often facilitates localization of the arytenoid, I believe that it increases the likelihood of injury to the mucosa when separating the capsule of the joint on its mesial aspect and also interferes with direct laryngoscopy as practiced by some surgeons to determine the width of the glottic chink at the time of operation.

While the choice of preoperative medication and anesthesia must be left to the individual operator, it is, nevertheless, imperative that no preliminary sedation or general anesthesia is employed in a dyspneic patient who has not been tracheotomized. Tracheotomy should always be done under local anesthesia without sedation. Local anesthesia has been employed in several cases for mobilization but has not been satisfactory, and either inhalation anesthesia or sodium pentothal are preferable.

A horizontal incision two inches long conforming to the skin creases of the neck is made over the wing of the thyroid so that it crosses the posterior border of the cartilage slightly below its middle. The omohyoid may be retracted or incised. The posterior border of the thyroid cartilage is identified and the inferior constrictor is incised along its attachment to the cartilage. The palpating finger should now identify the signet of the cricoid along its upper margin. As a rule, the arytenoid cannot be palpated due to its forward displacement.

The pharyngeal mucosa is separated for a short distance from the signet of the cricoid cartilage towards the midline and the posterior cricoarytenoid muscle is identified and incised along its atttachment to the muscular process. As a rule there is little difficulty in identifying and finding the muscular process of the arytenoid. I have found a small hook valuable to secure the arytenoid during these manipulations. In order to secure satisfactory mobilization it is well to separate all of the posterior cricoarytenoid muscle from the muscular process, to incise the fibres of the arytenoideus muscle as much as one can along the body of the arytenoid and then to pull the arytenoid backward to separate some fibres of the lateral cricoarytenoid muscle. The cricoarytenoid joint is opened and its attachments are incised. Care should be exercised to avoid injury to the laryngeal mucosa, particularly when separating the mesial attachment of the capsule. A blunt elevator has been found satisfactory for this procedure.

Before proceeding with the placement of the suture it is necessary to assure oneself that the arytenoid is freely mobilized. It should be possible with the aid of the hook to draw the arytenoid laterally and posteriorly so that it can be brought in contact with the posterior border of the thyroid cartilage.

Lateral fixation is accomplished by securing the arytenoid to the posterior border of the thyroid cartilage. For this purpose a full curved needle with a braided No. 0 silk suture is passed around the cartilage and secured through a hole made with a dental burr near the posterior border of the thyroid cartilage. This gives better fixation than if fascia or muscle is used. The suture should be placed around the arytenoid near its base, beneath the mucosa, passing the needle from within outward. Occasionally it is difficult to place the suture in proper position about the arytenoid since this cartilage is pyramidal in shape and the mucosa along its inner surface is in close relation to the perichondrium. In difficult cases the mucosa overlying the posterior and particularly over a part of the inner aspect of the arytenoid should be separated. Perforation of the overlying mucosa may lead to the formation of a granuloma or to infection of the wound. While it is desirable to pass the needle around the cartilage, I am sure that in many instances it is passed through a part of the cartilage along its inner aspect.

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The pharyngeal mucosa is separated for a short distance from the signet of the cricoid cartilage towards the midline and the posterior cricoarytenoid muscle is identified and incised along its atttachment to the muscular process. As a rule there is little difficulty in identifying and finding the months after operation, the airway does not appear wide enough and decannulation has not been carried out. A glottic chink should be approximately one-half cm. in width at its posterior end to insure an adequate airway.

End-Results: It is impossible to restore normal laryngeal function in a case of bilateral abductor paralysis. In order to improve the airway one must widen the glottis and this obviously will detract from the patient's ability to phonate. One, therefore, can state positively that the wider the glottis is, the poorer the voice will be. It has been observed that the operated side becomes fixed, and any movement that is observed occurs on the unoperated side; therefore, while one may operate on both arytenoids a unilateral operation is preferable from the standpoint of phonation. Better phonatory results also will be obtained if the arytenoid is retracted laterally without rotating it so that a tense straight-edged vocal cord will be secured.

I had previously believed that such adduction as occurred on the unoperated side could be explained by the action of the cricothyroid muscle which primarily is a tensor of the cord but also exerts some adductor action. When the cricoid cartilage is drawn upward towards the thyroid cartilage by the cricothyroid muscle, there apparently is some tilting inward of the corresponding arytenoid, which probably is a passive action. The thyroarytenoideus muscle by reason of its attachment to the vocal process also may act in a passive manner. There is considerable question whether the action of the cricothyroid muscle is of great significance in posticus paralysis. The arytenoid which is tilted forward lacks normal fixation and this interferes with the tensor action of the cricothyroid muscle. It seems reasonable that the unopposed lateral cricoarytenoid muscle may be responsible for slight adductor action on the nonoperated side. I believe that many of these patients use the extrinsic muscles which aid in narrowing the airway during phonation. In addition one must take into account that there is a change in the upper orifice of the larynx, for the epiglottis usually is tilted backward and on phonation this is increased contrary to findings in the normal larynx.

Voice Changes: In none of my operated cases has there been any material improvement in the voice. In practically all of the cases of paralysis following thyroidectomy the voice was quite clear before operation although there was breathlessness. In a majority the voice has been reasonably good following mobilization; there has been but one exception and in this the voice was a loud whisper. Patients who have worn a tracheal cannula for relief of dyspnea or have lived a precarious existence because of shortness of breath invariably are extremely grateful when the airway is increased. I have never observed a single instance in which the patient regretted having had the operation performed in spite of the voice impairment.

Statistical Data: To date I have operated upon 66 patients. In the first three and in Case 18 a second operation was necessary because the airway was not adequate. In the three the omohyoid was transposed and in the latter case there was edema of the arytenoid on the operated side which persisted. While there was no wound infection, there undoubtedly was some chondritis of the arytenoid cartilage and a second operation was performed with very satisfactory respiratory results and fair phonatory results considering that both arytenoids are immobile.

Sex: There were 56 women, nine men, and one boy aged 14 years.

Etiology: In 60 the paralysis followed one or more thyroidectomies. In three the cause was not known, but apparently was toxic or viral in origin. In one of these, a boy aged 14 years, bilateral paralysis followed measles seven years previously. In two there was trauma due to gunshot and shrapnel wounds, and in one there was bilateral paralysis following pulmonary tuberculosis.

Duration of Paralysis: In 34 cases the paralysis was of less than five years' duration. In 11 it was from five to 10 years; in 13, from 10 to 20 years, and in six it was over 20 years, the longest being 24½ years.

Age: The patients' ages varied from 14 years, the case of the boy with measles, to 76 years. Over one-half of the patients were more than 35 years of age. Twenty-eight at some time had worn a cannula; one wore a cannula for 16 and another for 17 years.

Decannulation Following Mobilization: In the earlier cases decannulation was delayed a number of weeks. Now, however, it is carried out more promptly as there has been opportunity to observe the postoperative reactions. This also may be due to the employment of antibiotics or to more careful handling of tissues. In 20 of the last 21 cases decannulation was completed within two weeks, the earliest being five days postoperatively. In one case the tube was not removed until after three months. This patient developed a small granuloma in the posterior commissure on the operated side that suggested a contact ulcer although it probably was the result of trauma to the mucosa. Removal of the granuloma was followed by complete recovery and decannulation with a reasonably good voice.

In two cases following thyroidectomy and in one post-traumatic case the cannula has not been removed. In one of these, Case 2, there was no lateral fixation of the arytenoid as the omohyoid was employed. The patient did not wish a second operation. In the second case, now two months postoperative, the glottis appears too narrow, but there was considerable submucosal hemorrhage at the time of operation. In the post-traumatic case decannulation was carried out, but because of extensive cicatricial changes subglottically in the posterior commissure there was recurrence of the stenosis, and tracheotomy again became necessary. This case was not suitable for this type of operative procedure.

Wound Infection: Infection of the wound due to injuries to the laryngeal mucosa occurred in five of the earlier cases. In only one was this protracted, but an ultimate satisfactory result was obtained in all.

While a majority of cases of posticus paralysis result from thyroidectomy, occasionally trauma to the neck may result in paralytic and cicatricial stenosis. It is not always easy to distinguish between these, nor can one determine preoperatively whether a satisfactory result may be secured.

Post-Traumatic Cases: In two cases, both veterans of World War II, there was a history of gunshot and shrapnel wounds of the neck and larynx with bilateral fixation of the arytenoids in the midline. The appearances were those one observes in post-thyroidectomy paralysis. In both, mobilization and lateral fixation of an arytenoid was carried out. The findings were quite unlike those observed following thyroidectomy in that there was fibrous ankylosis of the cricoarytenoid joint in one and bone ankylosis in another. The latter case, which was done more than 18 months ago, has retained an excellent airway and has been decannulated, but the patient with fibrous ankylosis had a recurrence of dyspnea about six months following decannulation and it was necessary to do a tracheotomy. On further inspection it was found that this patient had extensive cicatricial changes in the interarytenoid area, extending subglottically, which were not observed preoperatively and his case was not a suitable one for this type of operative procedure.

In conclusion, I believe that mobilization and lateral fixation of an arytenoid gives excellent results in bilateral abductor paralysis of the larynx. Other techniques have given similar results in the hands of various operators. The fundamental principles require separation of the cricoarytenoid attachments so that the vocal cord can be abducted and fixed in that position.

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STANDARDIZATION OF PURE TONE AUDIOMETER TESTING TECHNIQUE.*

SCOTT N. REGER, Ph.D.,
Iowa City, Iowa.

This paper presents: I — an enumeration of some of the factors which influence the accuracy of measurement of auditory pure tone threshold acuity; II — a brief discussion of the application of psychophysical methods to the determination of sensory thresholds; III — a review of the testing procedures usually employed in clinical pure tone audiometry; IV — an evaluation of audiometry as art or science; V — a description of a constant reproducible pure tone presentation technique which also indicates the mathematical probability of the accuracy of patient response; VI — a brief evaluation of two other types of audiometers (previously described in the literature) with which uniformity of tone presentation and highly objective analysis of patient response are attainable.

— I —

Among the factors which influence the reliability of pure tone audiometer test results are the following: *1.* the accuracy of the calibration of the audiometer; *2.* the magnitude of the masking effect of ambient noise in the testing environment, which includes noise produced by the audiometrist, the patient, and any sound in the receiver resulting from operation of the audiometer other than the desired test tone; *3.* position and pressure (coupling) of the sound reproducer in relation to the auricle or bones of the head; *4.* physical conditions within the test room which influence body comfort, such as temperature, humidity, barometric pressure, altitude,

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*Department of Otolaryngology and Oral Surgery, University Hospitals, State University *of Iowa, Iowa City, Iowa.*

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 19, 1949.

and time of day; *5.* age, intelligence, reaction time and previous test experience of the patient; *6.* physiologic condition and mental attitude of the patient (alert, interested, relaxed cooperative, indifferent, lethargic, depressed, fatigued, tense apprehensive or antagonistic). Patients who experience tinnitus or auditory after-images, malingerers and those with hysterical perceptual abnormalities present additional test difficulties; *7.* training experience, insight and personality of the audiometrist; and *8.* the complexity of the stimulus presentation technique used in the determination of the auditory threshold. Ambiguity of response instructions to the patient and the use of complex recording methods may be responsible for occasional errors in the patient's response or in the recording of the test data.

Westlake[18] has reported that the heavy "normal" line on the audiogram blank also is responsible for a common source of error. He found that if audiometrists give little or no attention to the decibel readings of the attenuation dial in relation to the zero reference level while testing, the results tend to vary from the "normal" line somewhat more than if they attend closely to the dial readings during the test. Brogden and Miller[3] have defined a "Physiological Noise Generated Under Earphone Cushions" as high as 25 to 30 db above the normal threshold value in earphones with small cavities, resulting from tremors of the muscles of hands and arms while holding the phones to the ears. This type of noise, of course, is minimized, if not eliminated, by the use of a well designed headband to hold the phones firmly in place against the ears. It is apparent that most of the above factors which influence the accuracy of measurement of auditory thresholds may be classified under the following three subdivisions: *1.* physical considerations; *2.* variables associated with the patient; and *3.* variables introduced by the audiometrist.

It is highly desirable that every audiometrist possess at least an elementary appreciation of the meaning of the term *threshold* or *limen*. Woodworth[10] states: ". . . It is a fundamental fact of psychology, and of physiology as well, that, *at any one moment*, a stimulus must reach a certain strength in order to be perceived or to elicit a response. . . . As its name implies, the threshold is a *transition point*. . . . The stimulus threshold is the transition point between stimuli too weak to elicit a response and stimuli strong enough to elicit that response."

Guilford[6] writes: ". . . There is no *single* stimulus value at any point on the scale below which a response never occurs and above which a response always occurs. There is a rather indefinite, twilight region of uncertainty, in which part of the time a response occurs and part of the time it does not. It is generally agreed that we should adopt that stimulus value which gives a response exactly half the time; in other words, the stimulus that has a probability of 0.5 of producing a response, as the *stimulus limen* RL. Above the RL, stimuli give a response more than half the time; below it they elicit a response less than half the time. . . ." Thurstone[17] points out that "The subject varies from moment to moment in his sensitivity and attention, so that it usually becomes necessary to define the limen as a statistical measure."

The absolute threshold of audibility of the "normal" ear usually is expressed in terms of the magnitude of the minimum sound pressure in watts or dynes per sq. cm. at a given point and frequency level required to elicit a response one-half the number of times it is presented. As is well known, clinical audiometers enable threshold measurements to be obtained in terms of decibels above or below the "normal" hearing zero reference intensity level, usually in multiples of 5 db intensity step intervals.

Myers and Harris[11] differentiate between ". . . a threshold of detectability and a so-called pure-tone threshold." Their data ". . . indicates that a zone of detectability of the order of 2 to 3 db exists between the intensity at which a tone is just detected 50 per cent of the time and the intensity at which it may be assigned a pitch quality of 50 per cent of the time." In a later article Myers and Harris[12] define this zone of detectability as follows: "This zone of detectability is defined as the intensity area between a 50 per cent detection threshold and 50 per cent pure-tone threshold." Pollack[13] also makes a distinction between "thresholds for the detection of the presence of a sinusoidal signal (thresholds of audibility and thresholds of tonality). . . . At each of the frequencies tested, the threshold of audibility was found to be significantly lower than the threshold of tonality. The difference between the two thresholds — the atonal interval — ranged from about 2.5 db at 500 cps. to about 6.5 db at 4,000 cps. Little difference was found between the variabilities of the two thresholds."

These studies by Myers and Harris, and Pollack indicate the advisability of differentiating between the detectability and pure tone thresholds while obtaining auditory acuity measurements, especially for research purposes. The observers must appreciate and experience the difference between the two types of auditory thresholds in a practice session and understand the exact nature of the stimulus to which they are to respond if pure tone threshold acuity data of the maximum possible accuracy are desired.

— II —

According to Chapter 12 of Garrett[7] since about 1829 considerable literature has developed around the subject of psychophysics, the branch of psychology devoted to the study of perceptual and sensory phenomena, with emphasis on the methods of determining sensory acuity and efficiency.

The psychophysical technique most suitable for the measurement of threshold acuity is the *method of limits,* also called the method of minimal changes or the method of serial exploration. The technique of the method of limits may be applied in the determination of auditory threshold values as follows: By means of a physically measurable and controllable sound source present the test frequency at a definitely audible intensity level and progressively decrease the intensity in small* intervals until the sound can no longer be heard — a descending series. Then reverse this procedure and increase the intensity until the tone can be heard—an ascending series. In theory, the transition point or threshold stimulus value lies between the last audible and the first inaudible presentation of the descending series or the last inaudible and first audible presentation of the ascending series; however, it has been found that the transition point in the descending series usually occurs at a slightly lower intensity level than the transition point in the ascending series. Consequently it is customary to regard the mean value of the last intensity level on the descending series and the first intensity level on the ascending series at which the observer O heard the tone as the threshold stimulus value. It is advisable to obtain response measurements on several pairs of descending and ascending stimuli for each O at each test frequency in order to obtain sufficient data for adequate statistical evaluation of the accuracy of the threshold measurements.

In order to minimize the errors of habituation and expectation inherent in the descending and ascending presentation of stimuli, it may be expedient to avoid the systematic progressive descending and ascending approach and present the stimuli in a haphazard or chance order. This procedure has been called the method *without knowledge*. For a detailed description of the psychophysical methods and their statistical analysis, see Guilford,[8] Woodsworth,[16] Thurstone[17] and Chapter 12 of Garrett.[7]

— III —

A review of the relatively few descriptions of audiometer testing technique in the literature suggests that audiometrists generally use an exceedingly abbreviated and modified form of the psychophysical method of limits for the clinical testing of auditory acuity. It may safely be assumed that these modifications are subject to considerable (if not infinite) variation in practice, since there seems to be no one generally accepted or standardized testing method. Consequently there are as many variations in exact procedure as there are audiometrists. Undoubtedly these variations in testing technique contribute in part to the well known observation that various audiometrists occasionally obtain significantly different results (audiograms) on a given patient with the same audiometer.

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*For the maximum attainable accuracy of measurement the discrete intensity step intervals should be slightly less than the intensity difference limen DL of the ear. The DL may be defined as the stimulus difference that can be perceived 50 per cent of the time. The sound intensity DL is a complex variable being a function of both the frequency and intensity levels at which measured. Riesz,[15] using a method of beats and 12 observers, found that at a sensation level of 5 db, the unilateral differential sensitivity varied from 2.48 db at 4,000 cycles to 9.32 db at 35 cycles. These data suggest that an intensity step interval of the order of 2 db is satisfactory for use in obtaining threshold data, although step intervals of 1 db or even less may be advisable in certain types of threshold measurements.

There appears to be ample justification for more or less modification of the classical psychophysical method for the clinical testing of auditory acuity. The psychophysical approach stresses the highest possible attainable accuracy under carefully controlled laboratory conditions. This methodology necessitates that the testing be done in a noise-free environment in terms of intensity increments that approximate a just noticeable difference (j.n.d.), and that a sufficient number of observations (responses) be obtained at each frequency level to amass sufficient data for significant statistical evaluation. The length of time required for the measurements and the statistical computation usually is of secondary consideration since the major emphasis is placed on accuracy of results.

In the average otologic clinic or office, however, a premium is placed on the minimum amount of time required to obtain measurements that are sufficiently accurate to indicate significant diagnostic information. The audiometer air conduction acuity measurements also may be augmented with bone conduction tests, some type of speech test, a test for recruitment and a test to indicate the presence or absence of stapes fixation, all of which are evaluated in relation to the history and physical examination of the ears, nose and throat of the patient. Under these circumstances the clinical audiometrist has been compelled to develop a technique by means of which significant hearing impairments could be detected with relatively little expenditure of time.

In the absence of a standardized test procedure it is not surprising that differences exist among the published descriptions of audiometer testing techniques. Bunch[1] recommends the following stimulus presentation technique (after instructing the patient to press a noiseless key every time he hears the test tone): "The sound should at first be loud enough for him to hear without any doubt. He should be told that the sound will be turned off and on and will become weaker and weaker until it can no longer be heard. As soon as these instructions are understood, the tone is presented through the receiver and the listener presses the key. The examiner interrupts the sound immediately, decreases the intensity and presents the decreased stimulus to the listener who responds as before. The process is repeated, the stimulus sound being made fainter each time until the listener no longer responds. After a few trials, a sort of rhythm can be set up. The stimulus is presented, the patient responds, the operator interrupts the sound, decreases its intensity and presents it again to the listener. This is repeated until the thresholds for all tones are determined."

This quotation indicates that Bunch favors a procedure in which the intensity is decreased systematically from audibility to inaudibility. Hughson and Westlake[9] recommend a presentation technique which emphasizes the increase of the intensity from inaudibility to audibility. "A reasonably loud signal should be presented first, basing the intensity on the apparent hearing loss. Attenuation is then introduced in 5 or 10 db steps until no response is indicated. Then take out attenuation until the tone is heard. This maneuver should be repeated and a uniform response obtained at least three times for each frequency involved. The interrupter button should be used whenever the attenuator dial is changed and the length of time the tone signal is presented should be constantly changed, thus avoiding any possibility of rhythmic response."

The writer dares add still another (and probably superfluous) description of pure tone audiometer tone presentation technique to the literature. The only justification for doing so is to present a detailed step-by-step procedure for beginning audiometry students to follow until they have acquired sufficient practice and experience to make modifications on the basis of their own experience. A basic understanding of the factors involved in accurate testing necessitates that certain threshold-seeking maneuvers be employed, which are used in common by all effective audiometrists.

During the testing procedure, it is advisable to rotate the attenuation dial only while the tone is suppressed by means of the tone interruption switch, if rotation of the dial results in perceptible contact noise or "static" in the receiver which cannot be eliminated by cleaning the contacts of the attenuator with carbon tetrachloride and lubricating them with a thin film of a high grade light oil. It is assumed that the audiometrist will know the intensity level settings of the attenuator at which contact noise stimulates the threshold of the normal ear at the different frequency levels generated by his audiometer.

A brief statement to the patient concerning the purpose of the test and an explanation of how he is to cooperate during the test constitute an important part of the procedure. The patient may be "briefed" as follows: "I want to find out how softly you can hear several different tones in each ear. We will test one ear at a time the better ear first, if you think you can hear better with one ear than the other. Each tone will be made comfortably loud at the beginning so you will know what it sounds like and will know what to listen for. Every time you hear the tone, no matter how loud or how soft, push down on this signal key to let me know you are hearing it. Push down on the key as soon as you hear the tone and keep it down as long as you can hear it. When you stop hearing the sound, take your fingers off the key and wait until you hear it once more before you push down on the key again. Since I want to find out how softly you can possibly hear the tone, be sure to push down on the key even when it is so soft you can just barely hear it." These instructions, of course, may be modified to conform to the apparent educational and intelligence levels of the patient. It is advisable that the patient indicate with a suitable signal the entire time interval *during which* the tone is heard for at least two reasons: *1.* to check on the patient's accuracy of response at or near threshold levels by varying the length of the time interval during which the tone is presented, as well as the time interval between successive presentations, and *2.* to enable the audiometrist to distinguish between patient responses to the sustained test tones and unintentional attenuator contact noise or other types of spurious intermittent noise.

For clinical testing, the writer believes it inadvisable to instruct the patient to differentiate between the threshold of detectability and the pure tone threshold as defined by Myers and Harris,[11] or the thresholds of audibility and of tonality as defined by Pollack.[12] (It is hoped that a uniform terminology will soon be adopted to designate the "awareness" and "recognition" thresholds defined by these investigators.) Distinctions between these two different sensory aspects of a given tone as its intensity is varied about the threshold, however, cannot be ignored in research studies concerned with highly accurate threshold determinations.

At the beginning of the test make certain that the audiometer attenuation dial is set at or near the zero (normal hearing) reference level before the earphones are placed over the patient's ears. Start the test at a frequency level of 1000 (or 1024) cycles per second. Proceed as follows: *Step 1:* Starting with the intensity level below the patient's threshold, increase the intensity of the tone about 10 db every two seconds by rotating the audiometer attenuation dial in the clockwise direction until the patient indicates that he hears the tone (which may be as high as 10 db or so above his actual threshold by the time the signal is made.) Note the attenuation dial setting or reading at which the patient indicated the sound was heard and present the intensity 10 db above this value for about three seconds so the patient experiences the tone at a comfortable listening and identification level. *Step 2:* Decrease the intensity below this level at a rate of about 5 db every two seconds by rotating the attenuation dial in the counter-clockwise direction until the patient releases the signal key, noting the level at which the key was released. *Step 3:* Increase the intensity above this subliminal level in 5 db steps every three seconds until the patient responds, again noting the level at which the patient indicated the tone was heard. (This second response to the sound usually is within 5 db of the patient's actual threshold value. Note that with three rotational movements of the attenuation dial, a clockwise, a counter-clockwise and a second clockwise rotation — a close approximation to the patient's threshold has been reached.) From this point on, the intensity of the tone is alternately decreased below audibility (as in Step 2) and increased to audibility (as in Step 3) until the patient responds consistently at a given minimum intensity level, 5 db below which there is haphazard or no response.

At least two and preferably three consistent responses at the same minimum level constitute a reasonably accurate indication of the patient's acuity for the frequency in question, assuming synchronous responses to the variations in the time intervals between tone presentations and the duration of the tones, both of which must be varied in succesive presentations in manually controlled audiometry.

If the patient appears to respond quickly and accurately at a given level, for example, 45 db, but slowly and somewhat haphazardly at the 40 db level, the audiometrist must adopt a "two out of three" or preferably a "three out of four" synchronized response requirement as the criterion of the correct threshold value for the frequency in question. For example, if the patient responds synchronously to three out of four presentations of the tone at the 45 db level but entirely haphazardly at the 40 db level, then the acuity would be recorded at the 45 db level.

If the patient's responses become increasingly erratic at near threshold levels, it is advisable to present the tone again at a level about 10 db above his apparent or approximate threshold to refresh his memory of the attributes of the tone for which he is listening.

It will be noted that the above approach to the threshold emphasizes the ascending or increasing intensity technique in which the intensity is gradually increased from inaudibility to audibility. A brief review of the entire procedure shows that the tone is presented alternately above and below the patient's threshold, progressing gradually from levels definitely above threshold to the threshold level. In Step 1, the tone is first presented at a comfortable listening level, in Step 2 is attenuated below audibility and in Step 8 is increased to five or possibly 70 db above the threshold. In succeeding steps the tone is alternately attenuated below audibility and increased in intensity in 5 db increments until a reasonably consistent response is obtained at a stable minimum intensity level.

Fortunately less time is required to obtain a threshold measurement as outlined above than is expended in reading the description of it. As indicated previously, the above procedure is merely one way of ascertaining the acuity for a given frequency. Undoubtedly threshold measurements of equal or possibly higher accuracy can be obtained with radical modification of the suggested technique. It is intended that the described procedure serve merely as a detailed guide for the beginning student of audiometry until he finds it expedient to make modifications on the basis of his accumulated experience. Needless to say, considerable modification of technique may be required to obtain reliable acuity measurements on young children, senile individuals and patients with hysterical or other types of perceptual abnormalities.

It is merely a matter of time until one or more of several nationally recognized professional groups or committees interested in the accuracy and reliability of audiometric measurements will recommend that certain aspects of manual audiometry be standardized. For example, general agreement on whether the auditory acuity should be ascertained by means of an ascending or descending intensity series (or a combination of both), the number of correct responses that should be required to indicate the threshold out of a given number of stimuli presentations at near the threshold level, and possibly the order in which the various test frequencies should be presented are questions about which there should be no uncertainty. Certainly the more uniform the test procedure, the less the confusion in the teaching and the practice of audiometry, with a resultant increase in the accuracy and reliability of test results.

— IV —

Unfortunately the belief is all too prevalent that since the audiometer is a so-called "precision" instrument designed for the scientific measurement of auditory acuity, the results of hearing tests made with the audiometer are correspondingly precise and accurate. Nothing could be further from the truth. Few will deny that most of the functional and operational features embodied in the modern clinical audiometer are based on considerable research and engineering skill; however, present day use of the audiometer leaves much to be desired from the scientific point of view. One of the most fundamental and elementary tenets of science is the application of an exact measurement technique under carefully controlled conditions. The "measuring stick" itself must remain an invariable factor and must be applied and evaluated in precisely the same way each time it is used. Its limits of accuracy (error of measurement) also must be known.

Due to the fact that the presentation of the test tones (the measuring stick) of the usual clinical audiometer is under the manual control of the audiometrist, there are as many variations in testing procedure (as many different ways of applying the yardstick) as there are audiometrists. As indicated previously, no two competent audiometrists could possibly test a given patient in precisely the same way, nor could a given audiometrist test the same patient twice in exactly an identical manner. If similar audiograms are obtained in either instance the skill of the audiometrist is principally responsible — not the naive assumption that since the audiometer is a precision instrument for the accurate testing of auditory acuity, hearing tests made with an audiometer are correspondingly precise. So long as every operational step in the testing procedure is dependent upon the manual control of the audiometrist, audiometry is and will remain principally an art rather than a science. Consequently an effective testing procedure can be acquired and applied only through practice. No one can make accurate hearing tests with the usual clinical audiometer until he is so familiar with the mechanical operation of the instrument and an adequate tone presentation sequence that his attention is focused on the patient's response rather than the manipulation of the instrument's controls during the testing procedure.

A second factor which supports the viewpoint that present day audiometry is more an art than a science is the fact that the evaluation of the patient's responses often is a matter of subjective judgment, especially when the patient makes frequent incorrect or haphazard responses during the testing procedure. When the response of such a "difficult" patient is synchronized with a tone presentation the audiometrist often wishes he knew if the response were merely chance coincidence or a significant response. If patients responded only during the presentation of tones and never made haphazard judgments, audiometry would be very simple indeed. It is a certainty that Bunch[4] was concerned less with the difficulties inherent in the mechanics of the operation of the audiometer than with the problem of the evaluation of the patient's responses when he wrote: "The reliability of an audiogram is directly proportional to the skill of the examiner."

The writer does not wish to foster the impression that the art of audiometry is inherently a grossly inaccurate and unreliable test procedure. A certain amount of skill necessarily always will be required to manipulate the controls of equipment which require frequent adjustment or attention from the operator. Obviously any decrease in the amount of manual control needed for the effective operation of a given instrument will result in more uniform and exact performance of that instrument. Audiometry will more nearly approach the scientific goal of measurement in proportion to the degree that the tone presentation technique attains invariable uniformity and the evaluation of the patient's responses becomes subject to quantitative verification.

— V —

It is obvious that a type of audiometer which would facilitate the *production of test tones in a predetermined reproducible sequence* or pattern, which did not present clues that would enable the patient to make correct responses when the tone was presented but not heard, and which permitted an objective quantitative evaluation of the patient's responses would possess certain definite advantages. Among these advantages are the following: *1.* Any given patient could be retested any desired number of times with an identical tone presentation technique; *2.* any desired number of individuals could be tested with the same invariable reproducible tone presentation sequence; *3.* if sufficiently simple to operate mechanically, an individual with very little training in audiometry could obtain results as reliable as an expert audiometrist and different audiometrists would obtain more nearly the same results on the same patient; and, *4.* if generally adopted, such an instrument would make possible the standardization of audiometer technique with increased universal accuracy of test results. Such an instrument is highly desirable from the research point of view. And to the extent that the tone presentation technique attained invariable reproducibility and the evaluation of the patient's responses approached objectivity, the testing of auditory acuity with such an instrument would be less an art and a skill and become a more scientific procedure.

An instrument constructed by the writer in 1944 meets the criteria suggested above in a reasonably satisfactory manner. As is often the case, since building this instrument, Gardner[6] pointed out that practically an identical design was suggested by Munson in a Bell Telephone Laboratories memorandum, which to my knowledge was not published in accessible literature until 1947. The first description of the mechanical tone interrupter devised by the writer is contained in a Master of Arts thesis by Bartell,[1] placed in the State University of Iowa

Medical School Library in August, 1945. Bartell[1] states: "The purpose of this thesis, then, is twofold: first, to describe a technique by means of which individuals can be tested and retested in an exactly reproducible manner, and second, to investigate the variability of normal bone conduction measurements in comparison to normal air conduction measurements. . . . The major apparatus consisted of an audiometer and an automatic stimulus interruption device."

The instrument consists essentially of a motor driven tone interruption device which can be attached to a commercial audiometer. Functionally, the gadget is used to produce one of four stimulus patterns as one, two, three or four dashes or tone pulses coincident with the presentation of an attention light. A quiet fractional horsepower motor is geared to rotate a shaft 6 inches long and 5/16 inch in diameter through one revolution every 10 seconds. Five bakelite discs, each 3/16 inch thick and 3 inches in diameter, are mounted rigidly on the shaft. A small leaf-spring switch is mounted near the periphery of each disc so that teeth-like projections, or the depressions between them, activate the switches to interrupt the test tones from the audiometer and turn on a signal light to indicate the interval during which the test tone will be presented as one, two, three or four pulses. The signal light disc (Disc No. 5) is cut with a 1/8 inch projection or tooth extending 6/10 of the periphery of its circumference (216 degrees). This projection closes a switch which turns on the signal light for a time interval of six seconds. The light, of course, is off the following four seconds. During the four second interval the light is off, the patient indicates the number of tone pulses heard and the audiometrist selects the desired frequency, intensity and pulse pattern which he desires to present next.

Disc No. 1 is cut with a depression 1/8 inch deep extending around 1/10 of its periphery (36 degrees) which presents the tone 8/10 of a second during a revolution of the disc. Disc No. 2 has two depressions of the same dimensions as that on Disc No. 1, separated 1/20 the circumference of the disc (18 degrees from each other). As Disc No. 2 revolves, the tone is presented as two pulses or dashes, each pulse being 8/10 of a second in duration, separated by a silent interval of one-half second. Discs 3 and 4 present three and four pulses, respectively, the dimensions of the projections and spaces between them being constructed to conform to the dimensions described for Discs 1 and 2. Each of the four pulse-patterns is phased on the shaft in relation to the signal light disc so that the signal light is always illuminated 0.4 second before a pulse is presented. The light is extinguished 0.1 second after the termination of the last pulse produced by Disc No. 4. A selector switch permits selection of the desired pulse pattern for each rotation of the rotor assembly.

Attachment of the mechanical tone interruptor to the audiometer does not disturb the frequency and intensity calibration of the instrument. The leaf-spring switches associated with the pulse pattern discs are connected in parallel with the tone interruption switch of the audiometer, which does not interfere with the manual operation of the audiometer tone interrupter switch; however, it may be necessary to modify certain circuit constants in the interrupter switch circuit of the audiometer to obtain optimum "attack" and "release" times at the initiation and termination of the pulses which are free from transient "clicks." A time interval of 0.1 second for both the attack and release of the pulses has been found satisfactory. The minimum duration of the "steady state" presentation of each pulse should exceed 0.2 second, since Munson[10] reports that: "Full loudness is not reached until over 0.2 second has elapsed." As indicated previously, the duration of the pulses of the present interrupter is 0.8 second. Further experimentation with the device is to be undertaken to ascertain the optimum relations between the duration of the pulses and intervening silent intervals in order to learn the minimum time required for reliable pulse pattern audiometry.

Very little more time is required to test the acuity by means of the pulse tone audiometer than is expended in manual audiometry. It may be found advantageous to combine the usual manually controlled audiometer tone presentation and the mechanically (or electronically) controlled pulse tone techniques in certain clinical applications as follows: first, determine the approximate threshold level according to one of the manual approaches described earlier; then employ the pulse tone presentation technique. The patient is instructed to listen for one, two, three or four tone pulses or dashes each time the light is on and to indicate the number of pulses heard immediately after the light goes off. Needless to say successive pulse patterns must be presented in random order.

The first pulse pattern may be presented 10 or 5 db above the approximate threshold. Each succeeding pattern is attenuated 5 db until either an incorrect or no response is obtained. Then increase the intensity of the next pattern 5 db. If the pulse pattern is heard correctly at this level, present a second pattern at the same level. If the second pattern also is heard correctly, attenuate 5 db and note the response. Determine the lowest intensity level at which at least two out of three patterns are recognized correctly. Due to the fact there are no defined criteria of correctness of response in terms of pulse tone audiometry, the writer, at the present time, arbitrarily regards the correct clinical threshold value as the lowest intensity level at which at least two of three pulse patterns are recognized correctly, assuming two out of two correct responses for the adjacent 5 db higher level.

As previously intimated, the technique of pulse tone audiometry includes an objective evaluation of the mathematical probability of the accuracy of the patient's response. In order to ascertain the probability of a repeated or combined event, the probabilities of the single events are multiplied. Assuming haphazard or chance presentation of any one of four different pulse patterns, the patient's chance of guessing correctly the single inaudible pattern is one in four, of guessing correctly two successive inaudible patterns is one in sixteen, of guessing correctly three successive inaudible patterns, one in sixty-four and so on. Application of the terms of the *binomial expansion* shows that the patient has nine chances in sixty-four (approximately one in eight) of guessing correctly any two inaudible patterns out of three, and 27 in 64 of guessing correctly any one pattern out of three. (For a discussion of probability statistics, see Guilford[8] or any standard text of statistical methods.)

Since the patient has an almost 50-50 chance (27 in 64) of guessing correctly one out of three inaudible patterns, the writer believes it is reasonable to require two correct responses out of three pattern presentations, in spite of the fact the patient has only nine chances in sixty-four (approximately one in eight) of guessing correctly any two inaudible patterns out of three inaudible pattern presentations. This requirement may seem excessively cautious in view of the fact that the auditory threshold is defined in terms of the minimum intensity at a given frequency level that has a probability of 0.50 of producing a response; however, as indicated previously, this 0.50 value is ascertained by means of a psychophysical technique which presupposes the presentation of a sufficient number of stimuli to amass sufficient data for statistical computation and analysis — a procedure requiring radical modification for clinical application. Additional clinical use and research with pulse tone audiometry are needed to determine the most effective testing procedures and to evaluate the usefulness of the pulse tone technique.

Pulse tone audiometry is ideally suited for certain research applications. For such use, it may be advisable to construct a table or key to indicate the intensity levels and pulse tone patterns to be presented at each frequency level. The observer's responses, recorded on an appropriate blank, constitute a permanent record for subsequent analysis. Various types of stimulus keys—ascending, descending or haphazard intensity series — may be devised to meet the requirements of the experiment. In any given key it is essential that the same number of one, two, three and four pulse patterns be used and that the order of presentation of the patterns be due to chance. Of course, keys with more or less than four different pulse patterns may be found advisable for certain experimental purposes.

One of the most effective uses of pulse tone audiometry is the utilization of this technique in the construction of a group pure tone test. Reger and Newby[14] found that reliable pure tone measurements can be obtained on school children down to and including the fourth grade. The group pure tone test detects the existence of high frequency losses which are not revealed by means of the group phonograph record speech (double digit) test.

Gardner,[6] in 1947, reported the results of several clinical threshold measurements as made with an electronically generated pulse tone audiometer. He found that ". . . the pulse-tone test procedure gives threshold results which are somewhat more reproducible than results obtained with the standard test procedure, although the time required to make the former is slightly longer. In terms of the attractiveness of the tests for the operator and for the listener, the pulse-tone test appears to be at an approximate three to one advantage. The normal threshold intensity calibrations of the two tests are in good agreement, so that either test may be used interchangeably with the other. For the very young subject, the pulse-tone test appears to be preferable in the majority of cases and tends to lower the age limit at which threshold measurements can be made. . . ."

— VI —

In addition to the mechanical or electronic pulse tone testing technique, at least two other types of pure tone audiometers have been described in the literature by means of which a uniform testing procedure is attainable. Brief functional descriptions of these instruments will be given at the present time to re-emphasize their desirable features and to bring them to the attention of those who may have missed the original articles.

The audiometer described by Burr and Mortimer[5] consists essentially of a discrete octave audiofrequency generator, an automatic random stimulus selector and a modified electrically activated typewriter by means of which "the patient's replies are automatically recorded on a card which forms without transcription part of the permanent record." A loud speaker is used for reproduction of the test frequencies in a free field. The test frequencies (125 to 8000 cycles per second in octave intervals) may be presented either as sustained pure tones or as so-called warble tones. "This is a tonal effect produced by varying the frequency over a small range about the mean frequency, the time of a complete cycle of this variation being one-thirtieth of a second." The audiometrist approximates the patient's threshold manually at the beginning of the test at each frequency level and adjusts the attenuator so that the threshold will lie within the intensity range of the test stimuli which are then presented by the automatic random stimulus selector. A signal light glows each time a test tone or a zero stimulus is presented. A "yes" button is pushed by the patient if the tone is heard, a "no" button if not heard.

The test tone is presented 30 times at each frequency level — five times at each of six different intensity levels — over a range of either 20 or 10 db. A zero stimulus is presented once, making 31 trials for the test at each frequency level. For example, assume that a preliminary manual test of a patient's acuity at a given frequency level indicates an approximate 40 db loss. The attenuation control of the audiometer is then adjusted so that the maximum and minimum intensity range of the 30 tone presentations will cover an intensity range of 50 to 30 db in 4 db steps — five times at a level of 50 db, five times at 46 db, five times at 42 db, five times at 38 db, five times at 34 db and five times at a level of 30 db — in random intensity order. (The tone also may be presented in similar fashion over a 10 db range in 2 db step intervals if desired.)

Each intensity level is correlated with a letter on an electrically activated typewriter key. Each time the "yes" button is pushed, the letter associated with the intensity of the test tone just presented is typed on the patient's record; each time the "no" key is pushed a semicolon is typed on the record. The maximum intensity at each frequency level — in this case 50 db — is assigned to the letter "a," which intensity level is indicated on the patient's record. Since pushing either the "yes" or the "no" button activates the random stimulus selector, thereby ending the present trial and initiating the next one, the rapidity with which the patient responds with the buttons determines the length of time required for the 31 presentations at each frequency level. According to the authors, this time is usually from four to five minutes; therefore, approximately 30 minutes are required for a test at seven different frequency levels.

The audiometrist analyzes the data typed on the patient's record and constructs a graph which plots the number of "yes" and "no" responses against the intensity level if desired. It is apparent that the testing procedure described by Burr and Mortimer conforms to the psychophysical method of limits in which the different intensity levels are presented in a chance or haphazard series.

The audiometer described by Bekesy[2] is also a self-recording instrument of the continuously variable frequency type which automatically sweeps through a frequency range from 100 to 10,000 cycles per second over a 15 minute interval. The frequency of the oscillator is varied by means of a motor which is geared to rotate a shaft which is attached to the frequency dial or control of the continuously variable audio-oscillator. Mounted on the shaft is a drum several inches in diameter on which is fixed (clipped) an audiogram blank. The patient is instructed to push down on a key until a sound can just be heard. As soon as the sound is heard the key is to be released until it disappears or can no longer be heard, whereupon the key is to be depressed again until the tone can be heard once more, and so on. Pushing on the key starts a second motor which drives back and forth in either direction the slider or contact arm of an attenuation network which covers an intensity range of 140 db in 2 db steps. Attached to the contact arm of the attenuator is an ink pen which records on the audiogram blank the position of the contact arm — the intensity levels — at which the continuously variable tone is heard. The audiogram blank for use with this instrument is specially constructed so that the intensity ordinates and frequency abscissas are correlated with the corresponding positions of the attenuator contact arm and the frequency dial of the oscillator. The recording or responses of the normal ear on the audiogram blank appears as a zigzag line, the median of which is a straight line which indicates the threshold acuity. After instructing the patient and turning on the equipment, the test requires so little attention from the audiometrist that it may be regarded as a self-administering and self-recording test.

The attention lights used in connection with the Burr and Mortimer and the mechanical and electronic pulse tone audiometers, and the varying frequency of the Bekesy instrument are devices which enable the patient to exert maximum attention during the presentation of the test stimuli. Conventional audiometer technique requires that the patient be listening intently with no relaxation of his attention during the critical part of the testing procedure, since he must not know just when a tone at or near the threshold level will be presented. Also he is attempting to indicate the instant the tone is presented and the instant it is terminated. This is expecting a higher order of attention over a longer period of time than can reasonably be expected. Fluctuation in the patient's attention is responsible for much of his erratic response and variability. If by coincidence he happens to be attending at the instant a near threshold level tone is presented, he may hear it; otherwise he may not. Stevens and Davis[16] make an interesting comment on a well-known psychological observation: "The inherent variability of the observer himself can be easily demonstrated by presenting him with a steady tone at an intensity very near threshold and requiring him to press a button during all the time that he hears the tone. Almost without exception observers press the button intermittently."

SUMMARY.

1. Many factors influence the accuracy of clinical audiometric measurements among which are the differences in tone presentation techniques used by various audiometrists and the difficulties inherent in evaluating the patient's responses.

2. Auditory threshold acuity measurements of the maximum accuracy are obtained by means of psychophysical techniques which necessitate securing a sufficient quantity of data under carefully controlled conditions to enable significant statistical analysis and evaluation of the data.

3. Due to "certain" practical considerations (such as limitations on time, personnel, equipment and testing environment), the testing techniques generally employed in clinical audiometry at best must be regarded as exceedingly abbreviated and modified versions of the psychophysical method of limits.

4. Variations in test results between different audiometrists will be minimized by the general adoption of a uniform tone presentation technique, including emphasis on the approach to the threshold (ascending or descending or mixed intensity series), the number of required synchronized responses to the stimulus at a minimum intensity level 5 db below which there is uncertain or no response, the sequence of presentation of the different test frequencies and possibly the duration of the response the patient is instructed to make to the audible test stimuli.

5. At the present time clinical audiometry is more an art than a science.

6. Although the audiometer is generally regarded as a precision instrument for the clinical testing of auditory acuity, the accuracy of the results obtained with the audiometer are limited principally by factors extraneous to the instrument itself.

7. Pulse tone audiometry introduces a testing procedure which possesses the following advantages: *a.* a method of retesting a given patient any desired number of times with an automatically controlled, exactly reproducible, tone presentation technique; *b.* a means of testing any desired number of individuals in an invariable manner; *c.* a technique by means of which a relatively untrained examiner can obtain results as accurate as those of the skilled audiometrist; *d.* the type of tone presentation makes possible an objective evaluation of the mathematical probability of the accuracy of the patient's response with relatively few data (responses); *e.* the stimuli are presented while the patient is alerted by a signal light to give maximum attention to the stimulus; *f.* there is little or no ambiguity of patient response; and *g.* pulse tone audiometry presents many feaures which can be standardized.

8. Pulse tone audiometry makes possible a practical group pure tone hearing test.

9. The audiometers described by Burr and Mortimer[5] and Bekesy[2] possess desirable self-administering and self-recording features, and constitute uniform testing procedures.

10. Standardization (general acceptance of uniform technique) of manual audiometry and/or of electromechanically controlled audiometers will introduce more science into the art of audiometry.

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OTOGENIC MENINGITIS DUE TO ESCHERICHIA COLI TREATMENT WITH CHLOROMYCETIN.*†

A. F. JUDGE, M.D., and W. L. DAVIS, M.D.,
Martinsburg, W. Va.

Escherichia coli meningitis continues to be a rare disease of interest mainly to the pediatrician since it occurs chiefly in the newborn. Schere,[1] in 1895, reported three cases in a series of 1,500 cases of meningitis in children. Craig,[2] in 1936, reported Escherichia coli as the etiological factor in half his cases of meningitis in children. Barrett, Rammelkamp and Worcester,[3] in 1942, were able to collect a total of 108 reported cases of Escherichia coli meningitis.

The disease is of interest to the otolaryngologist because otitis media and mastoiditis may be the source of infection leading to meningitis as described by Schere,[1] Vonderabe and Baurichter,[4] Thompson and Quinn,[5] and Segiet.[6]

Meningitis due to Escherichia coli was usually fatal until 1939, when Thompson and Quinn[5] in this country first reported the successful treatment of the disease with sulfonamides. Other recoveries following treatment with sulfonamides were reported in the next several years; however, treatment was usually prolonged and sequelae such as hydrocephalus and encephalopathy were not unusual.[7]

The introduction of streptomycin with its antibiotic effect on Gram negative organisms offered a new ray of hope in the treatment of Escherichia coli meningitis.

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*From the Department of Surgery, Baker Veterans Administration Center.

†Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 6, 1949.

Alexander,[8] in 1946, reported the successful use of streptomycin in the treatment of meningitis due to Escherichia coli. The patient was a 19-year-old soldier and the original site of infection was in the tibia. Streptomycin was administered intramuscularly for 16 days and intrathecally for 12 days, with an additional dose on the fifteenth day. The initial cell count in the spinal fluid was 507 cells, of which 70 per cent were polymorphonuclear cells. Spinal fluid cultures were positive for Escherichia coli on the first and fourth days. There was dramatic improvement in the patient's general condition, but the cell count in the spinal fluid remained about 200 until intrathecal treatment was discontinued.

Shields,[9] also in 1946, reported the successful treatment of Escherichia coli meningitis in a five-week-old infant. The patient was given streptomycin by both intramuscular and intrathecal routes for 10 days. The blood culture and spinal fluid culture both showed a pure growth of Escherichia coli. The initial cell count in the spinal fluid was 68 cells. The patient recovered without sequelae. At the time of discharge from the hospital, 19 days after admission, the cell count was 120 cells, mostly lymphocytes. At a check-up three days later, it was 154 cells. The spinal fluid culture became negative for Escherichia coli two days after the initial dose of streptomycin.

Lieberman,[10] in 1947, reported a third case of Escherichia coli meningitis successfully treated with streptomycin. The patient was a nine-month-old infant and the etiology was unknown. Streptomycin was administered intramuscularly for nine days and intrathecally for four days. The initial cell count in the spinal fluid was 760 cells, of which 90 per cent were polymorphonuclear cells. Further cell counts were unreliable as the spinal fluid was persistently blood tinged. The patient recovered clinically on the fifth hospital day and had no sequelae.

The introduction of chloromycetin furnished the physician with an antibiotic having five times the power of streptomycin to inhibit the growth of Escherichia coli.[11] When administered by mouth, the drug rapidly appears in the blood and urine;[12] it also appears in the spinal fluid in a concentration approximately half that noted in the blood, and would appear to make intrathecal administration unnecessary. Clinically, chloromycetin has shown no toxic effects. It is administered orally, and in comatose patients it may be given by gavage. It may be administered parenterally in a solution in proylene glycol, but such usage is not recommended. Because of its characteristics, chloromycetin was selected in treating the following case of Escherichia coli meningitis:

History: S. K., a 25-year-old white male veteran, was admitted to the Baker Veterans Administration Center, July 6, 1949. He complained of a chronically discharging right ear dating back to May, 1944, when he incurred a rupture of his right membrana tympani while in the gun turret of a naval vessel. Following this injury, the ear drained intermittently and the discharge had a foul odor. In addition, he had noted some loss of hearing in the right ear. Treatment, including the recent use of aureomycin ear drops, had failed to clear up the discharge. His general health had otherwise been good.

Physical Examination: There was a marginal perforation of the right membrana tympani in the posterosuperior quadrant and a moderate amount of foul-smelling pus coming from the middle ear. There was slight deviation of the nasal septum to the right and some lymphoid tissue in the nasopharynx. Physical examination was otherwise normal.

Laboratory Examination: Examination of the blood showed: hemoglobin, 13 gm.; red blood cells, 4.3 million; white blood cells, 8,500, with 65 per cent polymorphonuclears. The urinalysis was normal. The Kahn test was negative. X-ray examination of the chest showed no abnormality. X-ray examination of the mastoids showed clouding and sclerosis around the right mastoid antrum. The left mastoid showed no evidence of disease. Audiogram showed a hearing loss of 62 db on the right and 6 db on the left.

Clinical Course: On July 12, 1949, under general anesthesia (sodium pentothal), a right modified radical mastoidectomy was performed. There was moderate destruction in the region of the antrum with granulations and a membrane resembling cholesteatoma present. There was a pathological exposure of dura in the region of the tegmen tympani, but the dura appeared healthy. Culture of the mastoid antrum at the time of operation showed a growth of staphylococcus aureus, Alpha streptococcus and Escherichia coli colonies. Following operation the patient was placed on procaine penicillin, 300,000 units daily, as a matter of routine. On the second postoperative day, the patient's temperature was elevated to 103.4° F., and the patient complained of nausea and headache. Examination revealed no nystagmus, no Brudzinski sign and no Kernig sign. The patient was placed on penicillin, 50,000 units every three hours. The wound was dressed and all packing removed from the mastoid cavity. Temperature returned to normal and the patient's symptoms disappeared. On July 18, the patient again complained of headache and temperature rose to 101.6° F., with a corresponding rise in pulse and respirations. Blood examination showed a count of 13,600 white cells, of which 68 per cent were polymorphonuclears. The following day the rectal temperature was 104.4° F. and the patient seemed lethargic. Examination revealed some stiffness of the neck and the Kernig sign was positive. Spinal tap was performed and pink, opalescent fluid obtained. The pressure was elevated to 600 mm. Laboratory examination of the spinal fluid showed a cell count of 4,500, with 95 per cent polymorphonuclears. There was no sugar present. Total protein was over 200 mg. No organisms were seen, but a culture showed a growth of Escherichia coli. The patient was given 500 cc. of whole blood and during the next 24 hours he received 20 gm. of sodium sulfadiazine intravenously. Penicillin was increased to 500,000 units every four hours. On July 20, the temperature was still elevated to 103.4° F., and the spinal fluid cell count showed 23,300 cells, of which 96 per cent were polymorphonuclear leucocytes. Smear of the fluid showed a few Gram negative rods. With this report of Gram negative rods found on smear and the culture from the previous day showing Escherichia coli, the patient was started on chloromycetin therapy. An initial dose of 3 gm. was given by mouth and followed by 0.25 gm. every four hours. Sulfadiazine was discontinued because urinalysis on this date showed sulfa crystals and large numbers of red blood cells. Ten thousand units of penicillin were given intrathecally at the time of the second spinal tap.

Twenty-four hours after the start of chloromycetin therapy, the patient was less drowsy; however, the temperature remained elevated and there was still present stiffness of the neck. He was taking fluids well by mouth. Spinal tap at this time showed the total white count to have dropped to 6,400, of which 97 per cent were polymorphonuclears. Sugar was still absent from the spinal fluid. The dosage of chloromycetin was raised to 0.5 gm. every four hours. At this time it was decided to convert the modified radical mastoidectomy into a complete radical to remove any possibility of a focus in the middle ear feeding the meninges. This was accomplished on the following day. At operation a few granulations were found over the exposed dura, but there were little additional abnormal findings in the middle ear. The wound was left wide open and packed loosely with plain gauze. The surgery was performed under intravenous pentothal anesthesia, and 500 cc. of whole blood were given following the operation. Spinal tap on this day showed the total cell count to be 3,700 white cells, of which 95 per cent were polymorphonuclear leucocytes. Culture of the fluid showed Escherichia coli to be present still.

Following completion of the radical mastoidectomy, the temperature dropped and spinal tap the next day showed only 950 white cells, of which 91 per cent were polymorphonuclears. Sugar was still absent. No organisms were found on smear and no growth was obtained from culture.

By July 27 the temperature had been down to 99° F. for four days. The patient was greatly improved clinically and the stiffness of the neck had largely disappeared. Penicillin, which had been continued because of the initial laboratory report of streptococcus and staphylococcus organisms in the mastoid, had been discontinued three days previously. The patient was taking nourishment well by mouth. Spinal tap on this date showed a white cell count of four, of which two cells were polymorphonuclear leucocytes. There were no organisms found on smear or culture. Sugar for the first time was present to the amount of 22 mg. Blood examination showed 3.1 million red cells, 6,500 white cells and 10.5 gm.

of hemoglobin. Because of the blood findings and the patient's complaint of weakness, another 500 cc. of whole blood were given. Chloromycetin was continued but the dosage reduced to 0.25 gm. every four hours.

On Aug. 1, a plastic closure of the mastoidectomy wound was performed. Three days later, the patient's condition continuing satisfactory, chloromycetin was discontinued. In all, the patient had received chloromycetin for 15 days without toxic effect. The total dosage had been 33 gm.

By Aug. 19 the patient had gained six pounds in weight and the mastoid cavity was becoming covered with epithelium. The patient was given a 10-day leave. Audiogram at this time showed a decibel loss of 46 in the operated ear. This was a gain of 16 db in hearing acuity. At the termination of the leave period the patient was again examined. The radical cavity had become almost completely epithelialized, the hearing gain was maintained and there were no residual neurological findings. The patient was then discharged from the hospital.

COMMENT.

The course of events in this case would seem to indicate a spread of the infection from the mastoid cavity directly to the meninges. The culture taken at operation showed Escherichia coli in addition to staphylococcus aureus and Alpha streptococcus. The rise in temperature two days postoperatively probably heralded a thrombophlebitis of a dural vessel and the second elevation four days later, the beginning of the diffuse meningitis. Blood culture was not taken.

The fact that the meningitis developed while the patient was on penicillin therapy and the failure of the patient to respond to large doses of penicillin would eliminate this antibiotic as a factor in the cure of the Escherichia coli meningitis.

The paucity of additional findings at the time the modified radical cavity was converted into a full radical cavity seems to relegate the surgical procedure to a very minor rôle, if any, in clearing up the meningitis.

Under treatment with chloromycetin, the patient's condition quickly improved and the spinal fluid culture became negative for the Eschericia coli organism within 72 hours. The cell count fell from 23,300 to 950 in this same period. Since the patient showed no toxic effects from the chloromycetin, it is likely that larger doses might have been used in the beginning of therapy. The patient was kept on the antibiotic after the spinal fluid became normal because of the necessity of doing a plastic closure of the mastoidectomy wound. It was felt that the surgery might "stir things up," so the chloromycetin was continued until this was completed.

No determinations of the concentration of chloromycetin in the blood or spinal fluid were made.

SUMMARY.

A case of fulminating meningitis due to Escherichia coli in a 25-year-old veteran is presented. The clinical course and operative findings indicated the infection to be otogenic. The meningitis rapidly cleared under treatment with chloromycetin by mouth. Although chloromycetin was administered over a period of 15 days, there were no toxic effects. There were no sequelae.

TABLE I. EXAMINATION OF THE SPINAL FLUID.

Date	Cell Count	Polymorphonuclears	Sugar	Culture
July 19	4,650	95%	0	Escherichia coli
July 20*	23,300	96%	0	Escherichia coli
July 21	6,400	97%	9	Escherichia coli
July 22	3,700	95%	0	Escherichia coli
July 23	950	91%	0	No growth
July 27	4	50%	22 mg.	No growth

*Chloromycetin therapy started.

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NEUROFIBROMATOSIS (VON RECKLINGHAUSEN'S DISEASE) WITH INVOLVEMENT OF THE LARYNX. REPORT OF A CASE.*

PAUL H. HOLINGER, M.D., and LAWRENCE L. COHEN, M.D.,
Chicago, Ill.

Although neurofibromatosis (Von Recklinghausen's disease) is a relatively common tumor, it rarely involves the larynx. Review of the literature reveals seven cases previously reported. Neurofibromas of the larynx are benign and cause difficulty only when they invade a vital structure. In the case herein reported, symptoms of the disease began in early childhood but caused no untoward symptoms until the growth in the larynx became large enough to interfere with swallowing and respiration.

CASE REPORT.

J. W., a 27-year-old white male, was seen in the otolaryngologic clinic of the *University of Illinois Research and Educational Hospitals* in May, 1949. His chief complaint was intermittent dyspnea and dysphagia which lasted for six months. According to the history, tumors appeared on his body when he was a year old and increased in number and distribution to involve the entire body. One such nodule, removed when the patient was eight years old, on histologic examination proved to be sarcoma.

In the Spring of 1948 the patient developed a cough and difficulty in breathing; later, alarming choking spells occurred together with some dysphagia. Examination of the larynx elsewhere had revealed a mass in the larynx, and he was referred for treatment to the University of Illinois Research and Educational Hospitals.

Indirect laryngoscopy revealed a round, pedunculated yellowish mass about 2 cm. in diameter that seemed to arise from the left pyriform fossa. It moved back and forth in the glottis upon respiration.

General physical examination was negative except for numerous nodules over the patient's body and the laryngeal findings described above.

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*From the Department of Otolaryngology, College of Medicine, the University of Illinois, Chicago, Ill.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 3, 1950.

Direct laryngoscopy confirmed the findings shown by mirror examination. On palpation with forceps the mass was found to be rather firm. The configuration and motility of the cords were normal. An attempt was made to aspirate fluid from the tumor, but no fluid could be obtained, confirming the impression that the mass was not cystic. Tissue was then taken for biopsy and proved to be edematous mucosa, tissue consistent with a polyp.

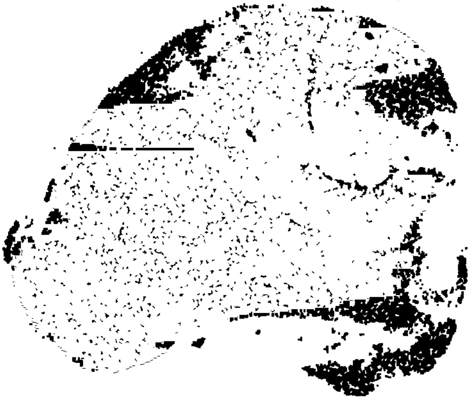

Fig. 1. Gross specimen of neurofibroma of the larynx measuring 3 x 2 x 1 cm.

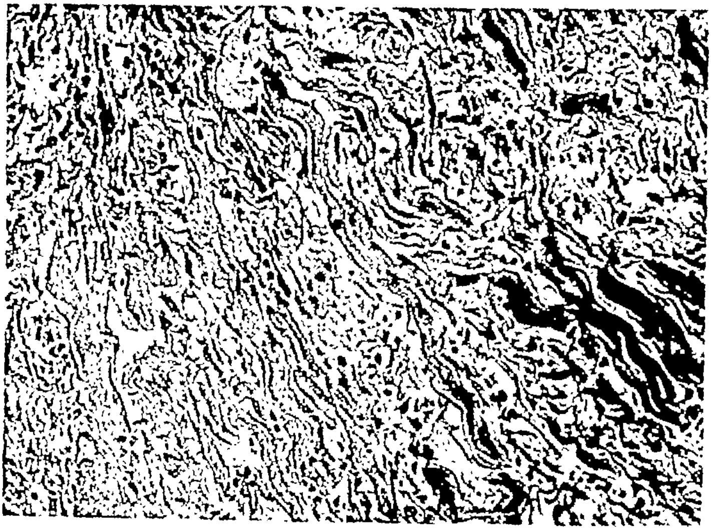

Fig. 2. Photomicrograph showing the characteristic appearance of neurofibroma (×430).

In the interim, one of the nodules removed from the patient's back proved to be a neurofibroma. Because of this, the mass in the larynx was suspected to be a similar tumor. A second direct laryngoscopy was performed several days later, and the mass was removed by a snare and laryngeal forceps with some difficulty, because of the firm fibrous nature of the lesion. Gross examination showed the tumor to be covered by normal appearing mucosa. Free bleeding ensued from the left pyriform fossa; this subsided rapidly, and the patient returned to the ward in good condition. Several hours later he coughed up bright red blood coming from the site from which the tumor had been removed. Unsuccessful attempts were made to control the hemorrhage by pressure under indirect as well as direct laryngoscopic guidance. Tracheotomy was performed; this allowed for more thorough packing of the pharynx and pyriform sinus, and the bleeding ceased. One thousand cc. of whole blood was administered and the patient was kept on intravenous feedings, strict bed and voice rest, and steam inhalation for a period of five days. The tracheotomy tube was removed on the seventh day, and the patient left the hospital in good condition. Histologic examination of the involved tissue established neurofibroma of Von Recklinghausen's type. Subsequent follow-up examinations have shown the larynx and pharynx to be normal.

DISCUSSION.

Von Recklinghausen's disease (multiple neurofibromatosis) is a relatively common condition,[1] but neurofibroma of the larynx is quite rare. New and Erich[2] report but one neurofibroma of the larynx seen during a 30-year period. Van Loon and Diamond[3] reviewed the literature and found descriptions of only six cases, to which they added one of their own. In only one of the seven previously reported cases of neurofibroma of the larynx was the laryngeal lesion associated with multiple neurofibromatosis.

In reviewing the present case, it is suggested that when removing a structurally similar pedunculated mass from the larynx, the base should be ligated or electrocoagulated.

SUMMARY.

1. A case of neurofibroma of the larynx associated with a generalized neurofibromatosis (Von Recklinghausen's disease) is presented.

2. Of the seven cases of neurofibroma of the larynx previously reported in the literature, only one was associated with the characteristic findings of Von Recklinghausen's disease.

3. Therapy consisted of snare and forceps removal of the laryngeal mass. A postoperative hemorrhage necessitated tracheotomy and pharyngeal packing.

4. There has been no evidence of local recurrence.

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MEETING OF GERMAN SOCIETY OF THROAT, NOSE AND EAR SPECIALISTS.

The annual meeting of the National Society of Throat, Nose and Ear Specialists will take place at Bad Kissingen under the direction of the president, Prof. Max Meyer, of Wurzburg, on May 24-27, 1950. The principal presentation will be "Mistakes and Dangers in the Treatment of Throat, Nose and Ear Diseases" by Prof. Marx, Wurzburg, with the collaboration of Prof. Moritz Weber, Ochsenfurth. American colleagues are heartily welcome as guests. Inquiries concerning invitations should be addressed to the secretary of the society, Prof. H. Frenzel, Geiststr 10, Gottingen, Germany.

UNIVERSITY OF MINNESOTA COURSE IN OTOLARYNGOLOGY.

The University of Minnesota announces the seventh biennial Continuation Course in Otolaryngology to be held June 26, 27, 28, 29, 30, 1950. This course is designed to bring to the practicing otolaryngologist the newer concepts and developments in the specialty. The course will be under the direction of Dr. Lawrence R. Boies and associates of the University Medical School. Dr. Fred A. Figi, Dr. Henry L. Williams and others of the Graduate School faculty will participate in the instruction. Guest lecturers will include Dr. Percy Ireland, Toronto; Dr. LeRoy Schall, Boston; Dr. Philip Meltzer, Boston; and Dr. John Shea, Memphis.

The fee for this course is $50.00. The enrollment is limited. Application should be made at an early date to the Director, Center for Continuation Study, University of Minnesota, Minneapolis 14, Minn.

BOOK REVIEW.

Theory of Hearing. By Ernest Glen Wever, Professor of Psychology, Princeton University. 584 pages with index and 137 illustrations. New York, N. Y.: John Wiley and Sons, Inc., 1949. Price $6.00.

This book is a technical monograph written by a psychologist for psychologists. It is a very personal book and sets forth the essence of the experimental work and theoretical interpretations by Prof. Wever and his collaborators over a period of 20 years. In it we find many details of the anatomy of the inner ear, and measurements of the size of many of the fine structures, and calculations based on these measurements that have not before been published. Here the book makes its most notable original contribution, and for this reason if for no other it will take its place among the classical treatments of the mechanism of hearing. Prof. Wever is undoubtedly the psychologist who is best acquainted with the anatomy of the ear. With his threefold experience in psychology, bio-acoustics and anatomy, he is able to make several unique contributions to our ideas of exactly how the ear receives sound waves and translates them into nerve impulses.

Prof. Wever's interest in anatomy is secondary to, and has its roots deep in his primary interest in auditory theory. His book is well named. Theories, not observations for their own sake and not practical applications, are his concern throughout. Prof. Wever and his colleague, Dr. Bray, are famous for their discovery of the electrical activity of the inner ear that is often known as the "Wever-and-Bray phenomenon," and they have put this phenomenon to good use as a tool wherewith to analyze many points about the bio-acoustics of the middle and the inner ear. Even their original experiment (placing an electrode in the auditory nerve of an anesthetized cat and "listening in" on the stream of nerve impulses going to the brain) was undertaken specifically to test a particular theory of hearing.

A central point in theories of hearing is the exact relationship between the pattern of nerve impulses and the pattern of the sound waves that stimulate them. Are the frequencies of sound waves carried to the brain as corresponding frequencies of nerve impulses? If so, does the brain make use of this information to recognize the "pitch" of a sound? If not, how does the ear analyze sounds in order to convey the information to the brain in some other form (such as sending the impulses over one set of nerve fibres rather than another)? The Wever and Bray experiment gave a partially affirmative answer to the first question. The frequency of the sound waves for low tones is in fact reproduced in the impulses in our auditory nerves. Prof. Wever has developed his theory of hearing, the "volley theory" as he calls it, to show that the brain might and probably does use these frequencies as an important clue for the recognition of pitch. He believes that his "volley principle" is important for all low tones and for medium tones up to almost the highest note on the piano.

If we can identify a plot or story in this book, we might call it "the rise and fall of the Helmholtz theory of hearing." Prof. Wever reviews in some detail the historical development of theories of hearing, and here again the book performs a useful service. Like Rosenblith and Békésy, who have also recently reviewed this subject, he makes it clear that the development of theories of hearing was limited at all points by the current knowledge of anatomy, physics and neurophysiology. The idea, formulated specifically by Helmholtz, that the newly recognized principle of "resonance" or sympathetic vibration might be the basis of a theory of hearing was fairly satisfactory in its day when less information was available than now. The principle of specific "tuning" by resonance was further developed by some of Helmholtz's followers, but it has few adherents today. Much of Prof. Wever's argument and many of his calculations are devoted to demolishing the remnants of the "Helmholtz" theory of specific isolated resonators in the inner ear. By the final chapter the reviewer had the impression that the author had been battling a straw man or perhaps a mummy, but Prof. Wever never relented!

Prof. Wever accepts the general present-day view that the inner ear does in fact analyze high-pitched sounds in a way somewhat like Helmholtz pictured, but for rather different reasons. The place or, better, the "place-pattern" of vibration in the inner ear is the essential feature. Some of us believe that the same general principle of place-pattern applies for all frequencies. Some may wonder why Prof. Wever passes so lightly over some of the evidence in favor of such an idea. The work of Stevens and Lurie, for example, is dismissed with hardly a word as apparently untrustworthy and not sufficiently detailed in presentation. The reviewer must protest this cavalier disregard of the work of his colleagues. In any case, Prof. Wever prefers a hybrid theory and invokes the frequency of nerve impulses and his "volley principle" as the chief cue for pitch for all tones below 1,000 cycles per second and as a partial cue for frequencies up to 4,000 cycles per second.

With Prof. Wever's final summarizing chapter the present reviewer can agree in full except for two or three sentences. The area of agreement far outweighs any difference of opinion on certain details such as the importance of the volley principle. Prof. Wever not only points out the difficulties of other theories, but he is also bold enough to formualte his own in full. In spite of some lack of clarity in the application of his theory to specific problems like tinnitus, diplacusis and binaural hearing, the main tenets of the theory are amply clear. Prof. Wever plays "doubles or quits." Within the next decade or two at the present rate of progress his theory may be as disregarded as that of Dr. Max Mayer, as outmoded as the formulations of Charles Bell, or so fully accepted that the rest of us will be saying that it is exactly what we believed all the time!

H. D.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1950.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 816 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) *Models* 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc.. 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-22, 1950.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Ralph A. Fenton, 906 Medical Arts Bldg., Portland, Ore.
Secretary: Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 23-24, 1950.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester, N. Y.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 25-27, 1950.

AMERICAN MEDICAL ASSOCIATION,
SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 17-20, 1950.
Meeeting: Palmer House, Chicago, Ill., October 3-6, 1950.

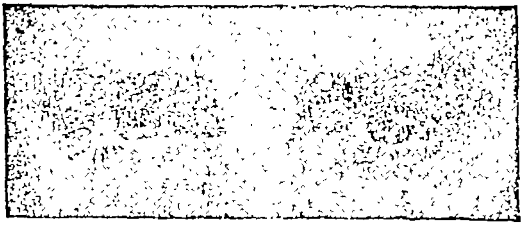
AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
President-Elect: Dr. Derrick Vail, Chicago, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 8-14, 1950.

How will they look to YOU a few years from now?

Your wife's eyes: What will you read in hers when she asks whether you can afford that modest cottage that's for sale?

Your boy's eyes: What will you see in his eyes the day he asks whether you can afford to send him to college?



Your own eyes: What will the mirror tell you about them when it's time to retire, and take things easier?

There's no better time than right now to sit back and think what *you* will see in your family's eyes a few years from now.

Whether they glow with happiness or turn aside with disappointment depends, to a very large extent, upon what you do *now*.

So plan *now* for that home you plan to buy eventually . . . set aside money *now* for his college education . . . plan *now* for the day you can retire.

Decide now to put part of your salary week after week, year after year in U. S. Savings Bonds, so that you will have the money for the *important* things you and your family want.

Insure your future by signing up on the Payroll Savings Plan where you work, or the Bond-A-Month Plan where you have a checking account. Chances are you won't miss the money now, but you certainly will a few short years from now if you haven't got it!!

Automatic saving is sure saving—U.S. SAVINGS BONDS

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THE
LARYNGOSCOPE.

VOL. LX MARCH, 1950. No. 3

BRONCHOSCOPY IN ATELECTASIS IN THE NEWBORN.*

A. W. DOUST, M.D.,
Syracuse, N. Y.

Bronchoscopy in atelectasis in the newborn has now become an accepted endoscopic procedure. During the past few years, the otolaryngologist associated with a general hospital has been called upon not infrequently by the obstetrician and pediatrician to aid in the treatment of newborn infants with respiratory distress. During this time the results have clearly shown that laryngoscopy and bronchoscopy of newborn infants are procedures which can readily be undertaken by the careful, trained operator without serious harmful effects to the patient. The operation is of both diagnostic and therapeutic value.

General Considerations:

Atelectasis is defined in Dorland's American Illustrated Dictionary as either an "imperfect expansion of the lungs at birth" or as "partial collapse of the lung." The word derives from two Greek words meaning "imperfect expansion."

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*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1950.

In medical literature there are four different conditions in the lung which are referred to as "atelectasis." The one most acceptable is fetal atelectasis, in which there is a failure of the lungs to expand at birth. The second type is the condition in which the lung is collapsed as a result of external pressure such as hydrothorax. The third is a condition known as "postoperative atelectasis"; the histopathologic aspects of which are inedaquately described because material is seldom available in the acute phase. The fourth is the condition in which the lung becomes airless and contracted after obstruction to the bronchus from an obstructive lesion, such as neoplasm.[1]

Physiology and Pathology:

Holt[2] states that in fetal atelectasis there is a persistence of the fetal state in the whole or in any part of the lung. The lung of the fetus is a uniform dark red color, and it shows lobular divisions very distinctly upon its surface. It is firm and solid, and sinks readily in water. Connective tissue is abundant and forms distinct fibrous septa which stretch across the lungs in every direction.

MacMahon[3] describes a separate pathological entity, congenital alveolar dysplasia. This is a morphologic anomaly of the lungs of newborn children. It is suggested that this anomaly represents a retardation and disturbance in the normal development of pulmonary alveoli. The etiology, at present, is obscure.

According to Brennemann,[4] in the intrauterine life the child's lungs are atelectatic. Twenty minutes after birth there is a 17 cc. capacity, and three to six hours after birth the capacity is 36 cc. Normally it is several days before the lungs have completely expanded. This expansion becomes complete first in the anterior borders and apices. The paravertebral, central and posterior portions are the slowest to expand. The inflation is sometimes irregular because of mucus in some of the bronchi.

Patterson and Farr[5] introduce strong evidence based on animal experimentation in support of the hypothesis that the human fetal respiratory tract is not inert in utero, but is subject to rhythmic respiratory movements, during which there is a tidal flow of amniotic fluid through the bronchial tree and alveoli. Examinations of lung secretions of neonatal deaths, some stillborn, showed amniotic fluid present. That this is not entirely due to passage through the birth canal, as is generally thought, is shown by the fact that some of these cases were delivered by Caesarean section. Snyder and Rosenfeld[6] showed that India ink injected into the amniotic sac is followed shortly by the appearance of this material in the pulmonary alveoli of the animal fetus.

Wilson and Farber[7] feel lack of expansion in the premature is not a failure of respiratory effort in many instances, though this may be weak, but that it is due to cohesion of moist surfaces of the air passages. This condition may, of course, be emphasized by any disturbance in the respiratory center, imperfectly developed thoracic mechanism or thorough obstruction of bronchi by aspiration of amniotic fluid, mucus or blood.

Incidence:

In an attempt to determine the incidence of pathological fetal atelectasis the number of cases so diagnosed in a general hospital of 340 beds was studied (see Table 1). In a period of five years (from 1944 to 1948, inclusive), it was found that out of 10,000 births there were 22 cases of fetal atelectasis, 15 of which expired and came to autopsy.

TABLE 1.

	Number of Births (Living)	Infants Deaths (Neonatal)	Still-born	Congenital Atelec-tasis
1944	1,700	39	34	4
1945	1,746	48	47	7
1946	2,100	55	55	5
1947	2,352	62	60	0
1948	2,202	33	67	6
Total	10,100	237	253	22

The postmortem findings are shown in Table 2. It is to be noted that in this series there were very few cases in which atelectasis *per se* was the primary cause of death. This is in accordance with the findings of Patterson and Farr,[5] who found in a series of 12 neonatal deaths that only five, or 42 per cent, were considered to have died from this condition. In the series here presented, six, or 40 per cent, expired from uncomplicated fetal atelectasis.

Ethel Dunham[8] states that "in cases of atelectasis in the newborn, failure of proper functioning of the respiratory center is generally held to be the most frequent cause of abnormal persistence of the fetal state in some parts of the lungs. Often the cause of the failure of the respiratory center cannot be determined, but immaturity, asphyxia, hemorrhage of the brain or edema may be responsible for the condition. Mechanical factors such as plugging of a bronchus by mucus or pressure on the lung by an enlarged heart or possibly by an enlarged thymus gland are said to have caused atelectasis of the lungs occasionally in the newborn infant. Atelectasis of the lungs in the newborn infant is therefore never primary but always secondary to some condition that interferes with the mechanism bringing about full expansion of the lungs."

Hemorrhage, edema, hydrocephalus, congenital malformations, bronchopneumonia, etc., were some of the contributing factors which appeared in this series. There was only one case in which variations in the Rh factor appeared.

It is noteworthy that in this group, eight, or 53 per cent, were prematures. Two were delivered by Caesarean section; and three were breech presentations.

The duration of life varied from approximately one minute to eight days.

Signs and Symptoms:

The classical picture of newborn atelectasis secondary to bronchial obstruction is, according to Owens:[9]

TABLE 2.

Case No.	Period of Gestation	Method of Delivery	Initial Respiratory Movements	Duration of Life	Gross Appearance of Lungs	Anatomical Diagnosis
(1)	Nine months	Breech difficult	Weak difficult resuscitation	Eight days	Expanded	Bilateral patchy atelectasis Incomplete development of adrenals Impetigo Mongolian (?) Hemorrhage and thrombosis of choroid in right lateral cerebral ventricle
(2)	Seven months	Spontaneous vertex	Weak	Twenty-three hours	Partial collapse	Prematurity Partial primary atelectasis Bronchopneumonia Neural thrombosis l. ventricle Subdural hematoma Extramedullary hematopotises
(3)	Seven months	Breech difficult	Weak	Twenty-four hours	Partial collapse	Prematurity Congenital atelectasis, bilateral
(4)	Six months	Vertex spontaneous	Weak	Twenty minutes	Massive collapse	Prematurity Atelectasis Hemaperitoneum Hematocoele Petechial hemorrhage in subarachnoid
(5)	Seven months	Vertex spontaneous	Weak: baby did not breathe for 10 minutes	Twenty hours	Partial collapse	Prematurity Cerebral edema Hydrocephalus Atelectasis
(6)	Eight months	Caesarean section	Weak	Thirty minutes	Massive collapse	Prematurity Congenital atelectasis (Mother Rh negative, Father Rh positive)

Case No.	Period of Gestation	Method of Delivery	Initial Respiratory Movements	Duration of Life	Gross Appearance of Lungs	Anatomical Diagnosis
(7)	Eight months	Spontaneous vertex	Weak	Twenty-four hours	Massive collapse	Atelectasis left lung and right lower lobe
(8)	Nine months	Low forceps vertex	Weak	One hour	Massive collapse	Atelectasis Congenital malformation of right diaphragm Diaphragmatic hernia (herniation: liver, small intestine, ascending colon and cecum through malformation)
(9)	Nine months	Caesarean section	Weak	Twenty-four hours	Massive collapse	Atelectasis left lung A subarachnoid hemorrhage
(10)	Seven months	Breech	Weak	Twenty-four hours	Massive collapse	Prematurity Atelectasis
(11)	Nine months	Spontaneous vertex	Weak	Twenty-four hours	Partial collapse	Bilateral pulmonary atelectasis Bilateral subarachnoid hemorrhage Dilated left ureter
(12)	Nine months	Spontaneous vertex	Weak	One hour	Massive collapse	Atelectasis, partial on right, complete on left Double kidney pelvis Anomalous ureters bilateral Congestion cerebral vessels
(13)	Seven months	Spontaneous vertex	Weak	Three hours	Massive collapse	Atelectasis Bronchopneumonia Neural thrombosis Subdural hematoma
(14)	Nine months	Vertex precipitous	Weak	Thirty minutes	Massive collapse	Congenital atelectasis
(15)	Nine months	Vertex spontaneous	Weak: two breaths only	One minute	Massive collapse	Congenital atelectasis

1. Progressive dyspnea with cyanosis, most marked after crying or other effort. This may often be temporarily relieved with oxygen. When these symptoms are not present, listlessness and pallor are usually noticed.

2. Suprasternal retraction with diaphragmatic tug on the lower ribs and diminished thoracic expansion on one or both sides, associated with suppressed breath sounds, with or without percussion dullness.

3. Coarse inspiratory rales and areas of localized emphysema. Coarse moist rales are an important finding and are usually absent in cases not due to body secretions.

4. Dehydration.

5. X-ray of the chest will usually show a rather complete atelectasis of one or more lobes. An X-ray should be made when possible in every case, but the diagnosis is made primarily on the clinical picture. The chest X-ray is also most helpful in distinguishing pulmonary from nonpulmonary entities.

Treatment:

The first requisite in resuscitation of the apneic newborn is the establishment of a free airway. Conservative measures such as catheter suction and external stimulation should first be tried. The major point to be emphasized with such stimulation is gentleness: the risk of causing visceral trauma, increased shock, and cerebral hemorrhage is very real. Oxygen is to be administered and warmth maintained.

Snyder,[10] in discussing the usefulness of respiratory stimulants such as alpholobeline, coramine, caffeine, metrazol and cyanide states that "it has been difficult to evaluate various reports concerning their rôle at the time of birth since these drugs are administered usually following labor, which is complicated by various factors that may alter respiratory activity, such as anorexia, anesthesia and trauma." In order to determine the effect of these respiratory stimulants upon the normal newborn, Lim and Snyder[11] carried out experiments on newborn rabbits immediately following delivery. Results showed that response by increase in rate or depth of breathing was brief, lasting less than a minute; furthermore, death frequently followed the onset of convulsions resulting from the administration of alphalobeline, caffeine or coramine.

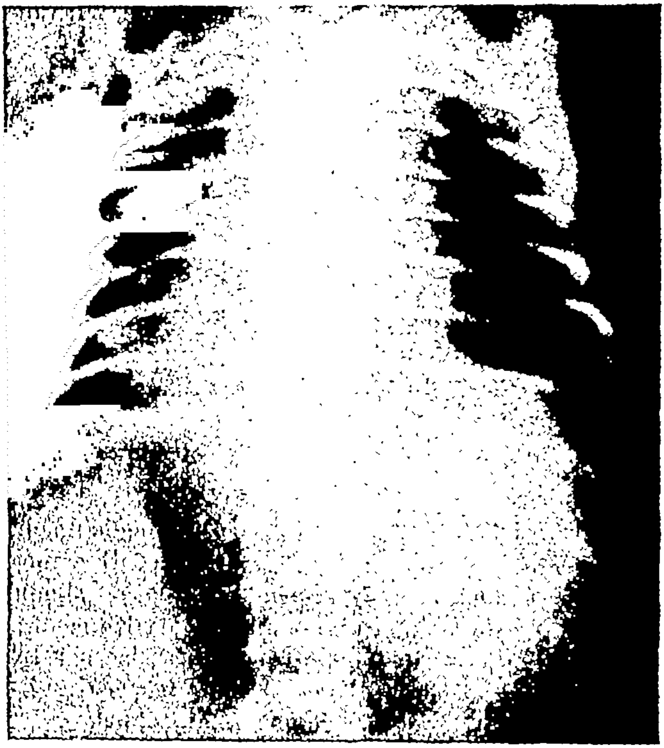

Fig. 1. X-rays from Case 1 showing fairly good ventilation in the lung fields with a wide heart shadow.

It has been the custom in some institutions to give vitamin K more or less empirically in cases of this kind in an attempt to control or modify hemorrhage. Fluids are to be given to control dehydration; and chemotherapy should be used to minimize the infection which usually accompanies prolonged atelectasis.

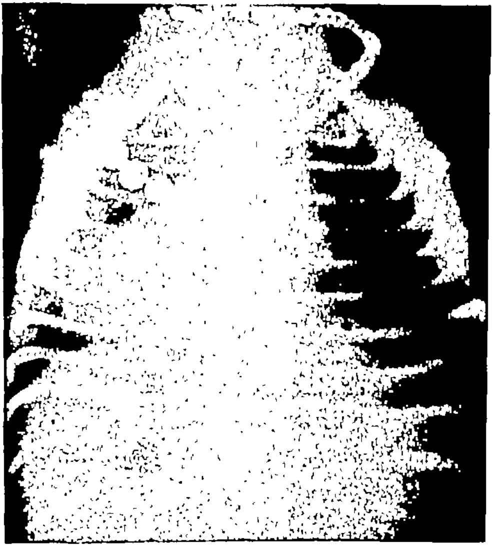

Fig. 2. X-rays of Case 2 with small area of atelectasis involving the posterior portion of the right lobe, with no shift of the central shadows.

Indications for Bronchoscopic Intervention:

Because of the great variations in the clinical picture, and rapid changes in the condition of atelectatic infants, final decision as to need for further treatment should rest with the bronchoscopist and pediatrician. The indication for bronchoscopic intervention, according to Owens,[8] is "any condition in which there is mechanical obstruction of the trachea or bronchus, not correcting itself spontaneously or with the use of approved conservative therapy, and which appears to be leading to the infant's exhaustion."

Following successful bronchoscopic aspiration, there is usually a progressive improvement in the infant's respiration; however, complete relief most frequently occurs six to eight hours after instrumentation.

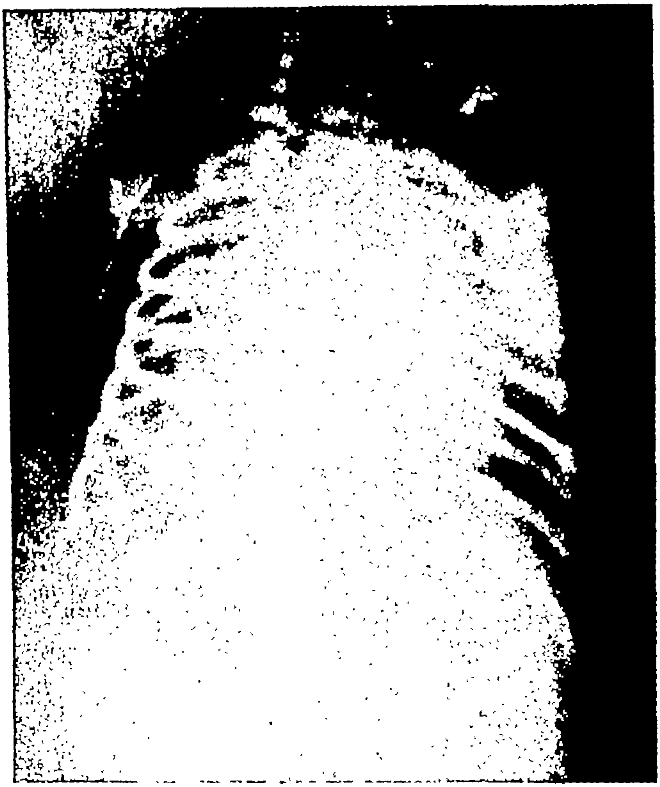

Fig. 3. X-rays from Case 3 showing bilateral atelectasis with very little ventilation on either side.

Case Reports:

The following six cases diagnosed as congenital atelectasis of the newborn have been bronchoscoped during the past eight months:

Case 1: Diffuse congenital atelectasis, from bronchial obstruction by body secretions.

A five-pound, one-ounce boy was born by Caesarean section. Because of dyspnea and cyanosis, it was placed in an incubator with an attached oxygen unit. Cyanosis appeared each time it was removed from the incubator. X-ray showed some fair ventilation of the lung fields, but the cyanosis and dyspnea persisted into the second day despite conservative treatment.

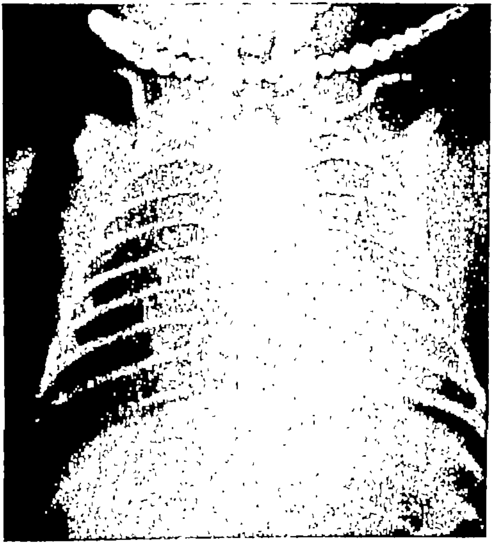

Fig. 4. X-rays showing massive atelectasis of the left lung with shift of the central shadows.

There being no improvement on the third day, an infant bronchoscope was passed. No marked obstruction was noted but a moderate amount of mucoid material was removed by suction. The entire procedure lasted approximately three minutes.

Symptomatic improvement was prompt and the child was discharged on the tenth day.

Case 2: Congenital atelectasis of the right lower lobe.

A six-pound, eleven-ounce baby boy, L. O. A., with episiotomy and low forceps, showed cyanosis and dyspnea with tugging. Placed in an incubator with attached oxygen unit, the cyanosis reappeared whenever the child was removed from the unit.

An X-ray of the chest showed a small area of atelectasis involving the posterior portion of the right lower lobe. There was no shift of the central shadows.

An infant bronchoscope was passed and suction applied to the right main bronchus. The entire procedure was completed in less than three minutes.

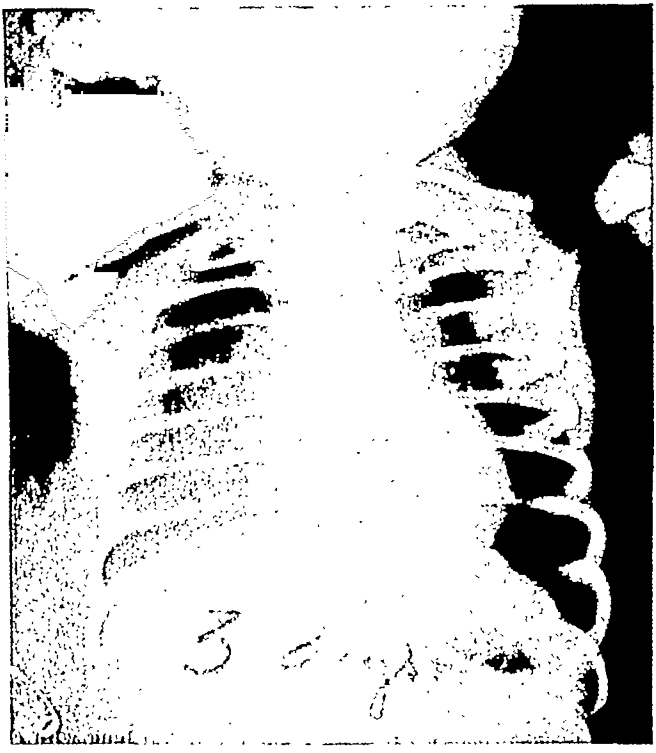

Fig. 5. X-ray showing appearance of chest three days after bronchoscopy.

Following bronchoscopy, the child's cry improved and the periods of cyanosis became less frequent. He was discharged on the tenth day.

Case 3: Bilateral massive atelectasis with bronchopneumonia.

A six pound, two and one-fourth-ounce boy, R. O. A., low forceps, developed rather marked cyanosis and dyspnea the second day. There was no improvement with mouth suction and frequent stimulation.

The child was placed in an incubator with attached oxygen unit, but little marked improvement was noted. An X-ray showed bilateral atelectasis with very little ventilation on either side. The heart shadow was large and indistinctly seen.

Two days following the onset of cyanosis and dyspnea, the child was bronchoscoped, conservative therapy having proved to no avail. Suction was applied and a moderate amount of mucus removed. No gross obstruction was seen.

There was no marked improvement after the bronchoscopic procedure, and the child expired 14 hours later. Unfortunately, no autopsy was obtained.

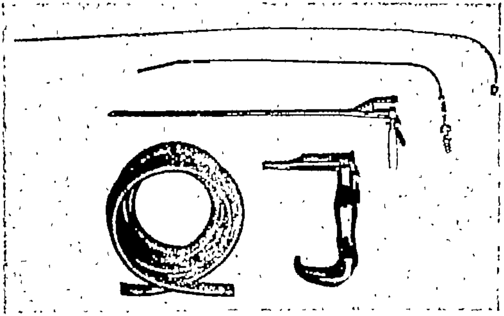

Fig. 6. Instruments used in bronchoscopic aspiration for atelectasis in the newborn. From above; infant suction tip, Samson infant aspirator, newborn bronchoscope, rubber suction tubing and newborn laryngoscope.

It was felt at the time that there was undoubtedly an accompanying central lesion, probably hemorrhage in this case; however, bronchoscopy was attempted without any apparent beneficial result.

Case 4: Fetal atelectasis, massive.

A seven pound, seven and one-half-ounce baby boy, L. O. A., born spontaneously, responded poorly to conservative methods of resuscitation, was bronchoscoped within two hours of birth. Large amounts of thick mucus were removed by suction.

Following bronchoscopy, respirations continued rapid and labored, but the color showed marked improvement. It was also noted in the physical examination that there was a supernumerary left ear.

The following day dyspnea and cyanosis were still marked and there was considerable tugging. Bronchoscopic aspiration was again helpful in somewhat relieving the cyanosis. At this time it was noted that the larynx showed no evidence of trauma from the previous manipulation.

Despite this second bronchoscopic drainage, the cyanosis returned and the dyspnea became more severe. The child expired from exhaustion approximately 14 hours after the second bronchoscopic procedure.

Postmortem examination showed both lungs smooth, glistening and maroon colored with no crepitation. It was of the consistency of liver. The bronchi contained a considerable amount of mucoid material.

An X-ray of the chest showed a small area of atelectasis involving the posterior portion of the right lower lobe. There was no shift of the central shadows.

An infant bronchoscope was passed and suction applied to the right main bronchus. The entire procedure was completed in less than three minutes.

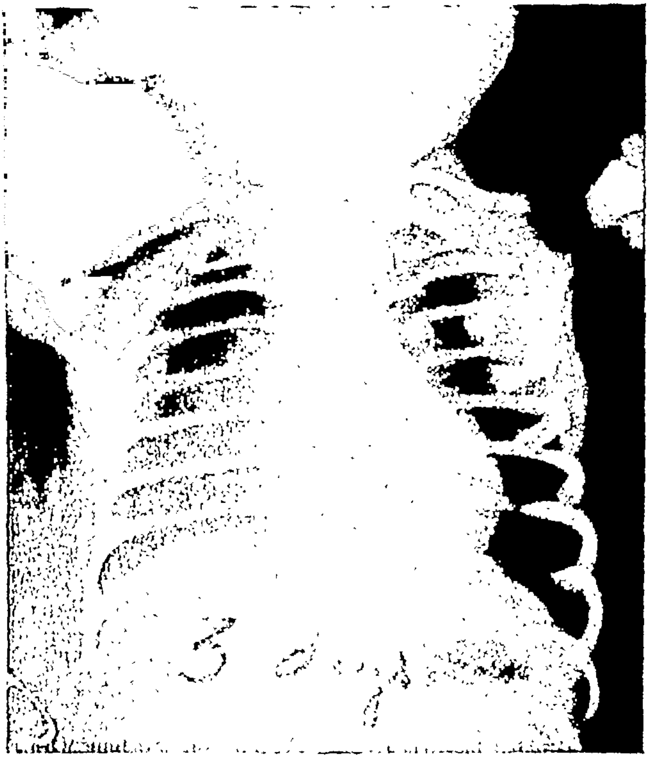

Fig. 5. X-ray showing appearance of chest three days after bronchoscopy.

Following bronchoscopy, the child's cry improved and the periods of cyanosis became less frequent. He was discharged on the tenth day.

Case 3: Bilateral massive atelectasis with bronchopneumonia.

A six pound, two and one-fourth-ounce boy, R. O. A., low forceps, developed rather marked cyanosis and dyspnea the second day. There was no improvement with mouth suction and frequent stimulation.

The child was placed in an incubator with attached oxygen unit, but little marked improvement was noted. An X-ray showed bilateral atelectasis with very little ventilation on either side. The heart shadow was large and indistinctly seen.

Two days later, at 8:00 A.M., the child's color was poor, lips cyanotic and respirations labored. The formula was gavaged at 10:00 A.M. His color continued poor, respirations became more labored, and since chest retractions were noticed, bronchoscopy was decided upon.

A moderate amount of mucoid material was withdrawn by bronchial suction. There was a minimal amount of trauma to the larynx and the patient's postoperative condition was good.

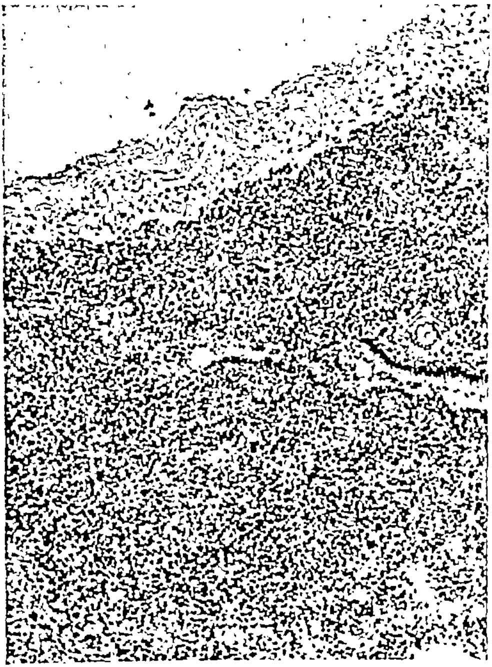

Fig. 8. Photomicrograph of atelectatic infant lung (pleura above).

At 5:00 P.M., the child was crying lustily and respirations were deeper. There was no chest retraction, and color was good. The following day the child showed marked improvement and no further bronchoscopic aspiration was necessary.

Comment:

Six cases of atelectasis in the newborn in which bronchoscopic aspiration was used as an adjunct to treatment have been presented. Two of the cases had a fatal outcome; however, in neither was there any evidence of harmful effect caused by the bronchoscopic procedure.

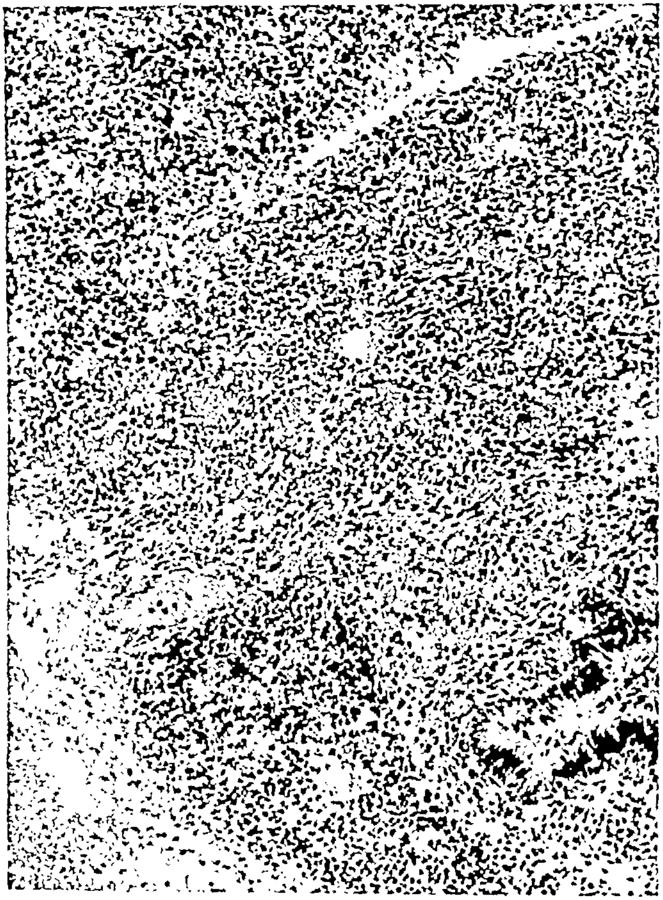

Fig. 9. Photomicrograph of normal expanded infant lung.

Very little equipment was necessary. The instruments used were the Holinger newborn bronchoscope, the Holinger newborn laryngoscope, the infant suction tip and the Samson infant aspirator. These are readily portable and may be moved from one hospital to another.

In each case an oxygen unit and sterile tracheotomy set were available for immediate use. Although extreme care was used in all cases in approaching and entering the glottis, in no instance did the entire endoscopic procedure consume more than three and one-half minutes. In two cases it was

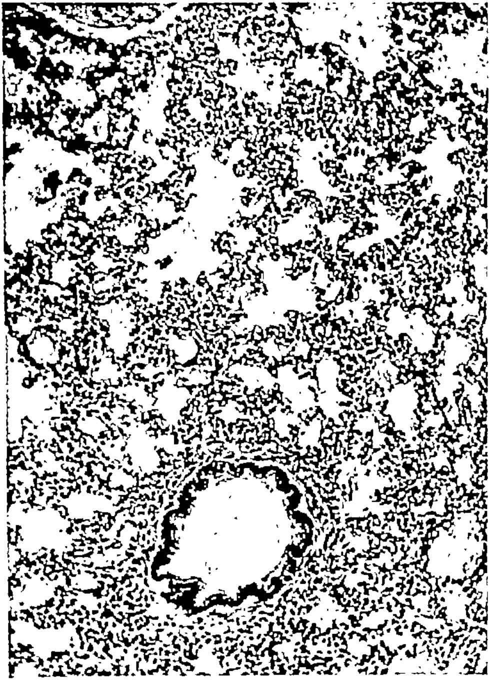

Fig. 10. Photomicrograph showing atelectasis and pneumonitis.

deemed necessary to give artificial respiration for several seconds before spontaneous respiration began.

It is most important to bronchoscope the infant before pneumonitis has a chance to develop in the atelectatic lung. Moersch[12] and others feel that such areas may perhaps be precursors of bronchiectasis.

Summary and Conclusions:

1. Bronchoscopic aspiration of the newborn is a relatively benign procedure when properly performed.

2. Selected cases of newborn atelectasis, secondary to bronchial obstruction, which failed to respond to conservative treatment, are materially benefited by bronchoscopic aspiration.

3. The etiology of atelectasis in the newborn is still not fully understood.

4. If bronchoscopic aspiration were more widely used for all types of congenital atelectasis, the indications and contraindications would be sooner understood and more lives undoubtedly saved.

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THE ROLE OF BACTERIAL INFECTION IN RESPIRATORY ALLERGY.*

LOUIS E. SILCOX, M.D.,
Philadelphia, Pa.

The otolaryngologist is so frequently confronted with the difficult problem of satisfactory treatment in cases of chronic sinusitis that an investigation into one of the important factors in its pathogenesis seems warranted. Most observers agree that between 70 and 80 per cent of all cases of chronic sinusitis have allergy as their basic pathology, and without adequate attention to this factor the treatment of this common disease process often ends in failure. The atopic or nonbacterial allergies have been exhaustively studied in reference to the respiratory tract,[1] but there is scant fundamental clinical data on bacterial sensitivity and the rôle it plays in the pathogenesis of chronic rhinitis, sinusitis and asthmatic bronchitis.[2-5]

Many clinicians are unwilling to accept hypersensitivity to infecting bacteria as an explanation of these conditions because the skin reactions obtained with bacterial products are of a delayed and inflammatory nature instead of the immediate urticarial type obtained with simple protein extracts. Some[6] believe that individuals possessing foci of infection, either in tonsils, teeth, sinuses or prostate, acquire a sensitiveness to bacterial protein which is similar to that of nonbacterial allergens, and that the infecting bacteria and their products alone and without the intervention of other foreign proteins may result in allergic symptoms. Billings,[7] over 80 years ago, was cognizant of focal infection as a cause of

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*Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1950.

anaphylaxis. He stated that the bacterial protein of the pathogenic micro-organisms of the focus may sensitize the body cells.

When a foreign protein—bacterial or nonbacterial—finds its way parenterally into the animal body certain changes take place in the body as a result of the contact with that protein. The exact nature of these changes is obscure, but the effects which they may produce if the same protein reaches the tissues again are quite familiar as the various hypersensitive reactions to foreign proteins. The reaction may come in the form of anaphylactic shock, asthma, allergic rhinitis, hay fever, serum sickness, etc.[8]

Zinsser[9,10] has pointed out that in allergy to bacterial infection we are dealing with the sensitization of the body by autolytically liberated antigenic substances which are absorbed from any focus in which bacteria react with inflammatory tissues, and as a result of which the body is subsequently rendered sensitive to contact with these same autolytic products, whether they are liberated and absorbed from a chronically existent focus or from an identical infection subsequently acquired.

Allergy to bacteria or their products is a phenomenon connected with many diseases such as tuberculosis, streptococcal infection, undulant fever, typhoid fever, etc., but best known in tuberculosis because of the discovery of the allergic phenomena in the skin of those who were infected by the tubercle bacillus.

That allergy takes part in the specific defense of the organism against a variety of substances, both bacterial and nonbacterial, is definite.[11] In tuberculosis after the body cells have once been sensitized by previous infection with tubercle bacilli allergy of varying degree, from a slight hyperemia to a severe exudation, may be met with whenever reinoculations of bacilli or bacillary protein takes place—the degree of allergy depending upon the dosage and virulence of the bacilli and the reacting capacities of the host. Such allergy is a specifically acquired reaction and is usually considered as part of the host's protective mechanism.

Two different types of immunologic reactivity were demonstrated by Zinsser.[9] Both the anaphylactic and the tuberculin types of sensitization were produced in the same animal. The "anaphylactic" type of reaction appeared in from two to 15 minutes after intradermal injection of the antigen. It expressed itself in the development of a growing wheal often surrounded by an areola. This reaction may last from one-half hour to one or two hours and fades without leaving any trace of injury to the tissue. The "tuberculin" type of skin reaction showed no immediate effect, but within four or five hours a swelling becomes apparent and in the course of 12 to 24 hours there is a swollen edematous area of varying intensity, often with a central necrotic spot and occasional hemorrhage. This reaction may not reach the height of development until 48 hours after the injection, and is accompanied by distinct signs of inflammation and some cell death.

The anaphylactic response on the part of the body to bacteria or their products is comparable in all respects to the nonbacterial sensitization, and the tuberculin type allergic response may be caused by any bacterial organism. Some inflammatory reactions of the body are necessary for its formation. Circulating antibodies are not a significant feature in the tuberculin type but definitely are in the anaphylactic type.[11]

If an animal is immunized with whole bacteria—either living, formalinized or killed in some other manner that avoids dissolution of the cell substance—we obtain antibodies which are type specific and bring about the well known test-tube reactions of agglutination, precipitation, opsonization, etc. If the bacteria are subjected to even the most gentle chemical cleavage the bacterial antigen may be split into at least two antigenically functioning fractions: one, a nitrogenous nucleoprotein-like material, the other a partial antigen, probably of carbohydrate structure which has been called the "soluble specific substance" by Avery.[12]

Bacterial allergy may be defined as a condition in which the body is sensitized to a bacterial antigen and this antigen in its most potent form results from biological disintegration of the bacteria, either by autolysis or similar disintegration which takes place in inflammatory foci; and it is likely that many of the experimental difficulties that have been encountered are due to the diminution of functional properties coincident to chemical manipulation *in vitro*. A formidable obstacle in explaining the specificity of bacterial effects lies in the extreme complexity of bacterial antigens and antibodies as compared with those of the simpler coagulable proteins.

The allergic state represents an increased capacity on the part of the tissues to react to this antigen, which may either come to the body in the form of a new infection or through the lighting-up of an old focus. This increased capacity to react may be injurious and result in pathological changes and disease.

The Arthus phenomenon is a special type of accelerated allergic response in rabbits and other animals. The Schwartzman phenomenon is an unexplained skin response to bacterial filtrates. These reactions are effects coming from bacterial metabolism and the corresponding complexity of reactions on the part of the host. It is entirely conceivable that some of these specific and nonspecific bacterial agencies enact an appreciable part in the pathogenesis of hyperplastic sinusitis, infectious asthma and other hypersensitive clinical states attributable to bacteria. It is evident that there are several types of allergy which differ particularly in the location of the antibody. In protein anaphylaxis the antibodies are in the cells in large numbers and are similar to those present in the sera. Their location and concentration are the factors which determine the suddenness of the reaction. In bacterial allergy the antibodies are also in the cells, but they arise from the activity of the infectious process and depend upon a third factor. In immunity they are almost exclusively in the circulating blood. Skin reactions are small, the resistance to infection is high and their concentration in the serum is the most important factor.

Whenever we find allergic phenomena in the course of bacterial disease we must assume that the reaction is due to some substance or substances produced during the growth of the causative micro-organism or derived from their intimate contact with the patient's cells after they have been acted upon and sensitized by a previous identical infection. The result of this contact is an inflammatory response.

There is no doubt but that bacterial infection of the respiratory tract may cause hyperplastic sinusitis and asthma. The latter may be acute paroxysmal or the chronic form. There have been some doubts as to whether hyperplastic sinusitis and asthma of infective origin can be considered as allergic reactions. This problem has been reviewed by many students of the subject and controversy still exists.

Some of the evidence in favor of an allergic response to bacteria or their products is the presence of an increase in the number of eosinophiles locally and in the circulating blood. Cooke[14] has reported an eosinophilic infiltration in hyperplastic rhinitis and sinusitis, and in a series of 131 active cases of asthma he found that a blood eosinophilia is not only more frequent but it is also on the average higher in infective asthma than in atopic asthma. He feels that eosinophilia of the tissues and secretions of sinuses and bronchi is an allergic reaction and cannot be due to the bacterial infections alone. Campbell[15] states that eosinophilia represents a cellular response which invariably accompanies local anaphylaxis and may or may not be evident in the general circulation, depending upon the severity of the hypersensitive reaction. Substances, such as adrenalin, which diminish or prevent anaphylaxis also diminish or prevent eosinophilia.

That the eosinophiles of the sputum in asthma and antral exudates in allergic sinusitis are probably of local tissue origin and not myelogenous is indicated by the fact that 15 cases with normal differential counts of the peripheral blood showed abundant eosinophiles in the secretion or exudate of bronchial and nasal mucous membranes and careful study of smears showed that the eosinophiles of the exudates are not all morphologicaly similar to those of the blood taken at the same time. Hyperplastic sinusitis and infective asthma may be regarded as an allergic reaction quite as properly as is the skin sensitive type—although the immunologic reactions in the two forms are not identical.

It is a well known fact that bacteria may be present in the sinuses or sinus mucosa in the absence of apparent infection.[16] The streptococcus is not a pus producing organism but tends to create edema and carry on in a subacute or chronic form. Virulent streptococci have been cultured from nonsuppurating hyperplastic sinusitis. Schenck[17] makes the observation that allergic ethmoidal tissue cannot combat infection effectively, and chronic infection results which favors bacterial sensitivity.

Cooke[18] states that almost 100 per cent of the pathologic manifestations of sinusitis are of the hyperplastic or thickened membrane type. Eighty per cent of the membranes he cultured were positive for one or more organisms.

A true hyperplastic membrane is much thickened, often infolding and polypoid with desquamation of the cilia, a much thickened hyalinized basement membrane and in the tunica propria marked edema, glandular hyperplasia, proliferation of the connective tissue and eosinophilic, round, and plasma cell infiltration. In the chronic cases neutrophiles are not frequent.

Grove and Cooke[18] felt that hyperplastic sinusitis is caused solely by infection which produced a special pathologic response conditioned by the allergic constitution of the patient. Grove and Farrior[2] found bacteria in 80 per cent of the ethmoid and sphenoid membranes at operation, and 96 per cent of the maxillary sinus membranes showed bacterial growth. The staphylococcus was the predominate organism and the hemolytic streptococcus was found twice as often in the antral membranes as in the ethmoid and sphenoid membranes. These allergic patients all had chronic hyperplastic sinusitis.

A comparison of organisms obtained from 87 maxillary sinus membranes with those grown from previous washings of the antra showed 43.7 per cent were different from any recovered preoperatively. Stained sections of tissues removed from the sinuses demonstrated bacteria very definitely in 85 per cent. The bacteria recovered *from 108 antral membrane* cultures were as follows:

Staphylococcus	57%
Streptococcus viridans	43%
Pneumococcus	39%
Hemolytic streptococcus	25%

Baird[19] raises the question as to whether the major allergen in house dust might be bacteria. Extensive studies show that one of the largest elements in household dust is bacteria, especially staphylococci and streptococci, and it is interesting to note that bacteria are practically the only allergens which increase in quantity after reaching the body.

The preceding theoretical and practical evidence as well as the success of autogenous vaccine therapy in selected cases of bronchial asthma by the author and others[3,19,33] led to this study of 110 patients with allergic hyperplastic sinusitis. There was reason to believe that the infectious element was the predominant factor in producing their symptoms, although two-thirds of the patients showed positive skin tests to non-bacterial allergens. Over one-half of the cases had bronchial asthma as well as allergic sinus disease. Sixty-seven had *hyperplastic mucosal changes without evidence of pus formation* in one or more paranasal sinus as evidenced by trans-illumination, X-ray or at operation, and 35 had gross pus formation in at least one sinus in addition to the hyperplastic changes. Ten patients had what appeared to be normal sinus mucosa, but *virulent organisms were cultured from all of* them.

The respiratory mucosa of the upper and lower tracts have much in common and it is logical to assume that if asthmatic attacks could be controlled by reducing allergic responses in the pulmonary tract with vaccine therapy, then many of the allergic reactions in the sinus mucosa might be reduced to a minimum if the proper organism could be obtained for antigenic activity. If these allergic edema reactions could be controlled or minimized, the fundamental pathology of most chronic sinusitis would be checked.

Cultures which were taken with due precaution to prevent contamination were obtained from the middle meatuses of the nose in 80 patients, from bronchoscopic aspiration in eight, from tonsil tissue at operation in eight, from sinus membranes at operation in seven, from maxillary sinus irrigation in four and from the nasopharynx in three.

TABLE 2.

INCIDENCE OF BACTERIA CULTURED FOR USE IN VACCINES.

Hemolytic staphylococcus aureus	42
Steptococcus viridans	34
Pneumococcus (Types 12, 31, 9, 4, 5, 3, 22, 23, 14)	29
Nonhemolytic staphylococcus aureus	29
Nonhemolytic streptococcus	27
Neisseria catarrhalis	19
Nonhemolytic staphylococcus albus	17
Hemolytic streptococcus	16
Hemolytic staphylococcus albus	13
Diphtheroids	3
K. pneumoniae	1
Proteus	1
Total	231

Castellani, in 1905, showed that when two organisms were injected into an animal simultaneously, the antibody response to each organism was equal to that produced by injection of the individual organisms. A good general principle to follow, however, in the preparation of mixed vaccines is that they should contain as few different bacteria as possible, and only those should be incorporated in full dosage which are known or reasonably suspected to be pathogenic. The ideal method of preparation of an autogenous vaccine is one which modifies the organism as little as possible — *i.e.,* just enough to rob it of its disease producing qualities without destroying its immunizing capacity.

TABLE 3.

VACCINES.

Pure culture		20
Hemolytic staphylococcus aureus	11	
Hemolytic staphylococcus albus	5	
Streptococcus viridans	2	
Pneumococcus	1	
Nonhemolytic staphylococcus aureus	1	
Two organisms in vaccine		57
Three organisms in vaccine		29
Four organisms in vaccine		4

Vaccines were prepared for this study by the following method: A sterile swab of the secretion from the selected site was inoculated into nutrient broth made from brain heart infusion, a blood agar plate, and a modified chocolate agar plate. The latter was incubated in an atmosphere of 10 per cent carbon dioxide. Colonies of pathogens were picked from the blood agar plates and smears were made of these, and the growth in the broth, for identification. They were then transferred to nutrient broth from the agar plates and were grown separately for four to five days.

The suspensions were filtered through sterile gauze, after transfer to a sterile flask. The filtered suspension was centrifuged and the supernatant fluid was poured off. Saline was added to each tube and a dilution of two billion per cubic centimeter was made using McFarland nepholometer tubes for comparison. The cultures were then mixed proportionately so that the final concentration of the mixture was two billion per cubic centimeter.

The mixed saline suspension was then poured into small vaccine bottles. A second bottle was prepared to a dilution of one hundred million per cubic centimeter. These were submerged to the neck in a water bath at 60° C. for one hour on two successive days. Tricresol was added to a final concentration of 0.25 per cent and 1 cc. from each bottle was transferred to brain heart infusion broth for sterility.

Intradermal tests were done routinely with 0.05 cc. of the weaker dilution.

The problem of skin testing with bacterial extracts is a difficult one. There are divergent views on this subject, and the percentage of positive results reported varies widely. Some authorities[24] state that many nonallergic persons and a high percentage of patients suspected of bacterial allergy give cutaneous reactions to a wide variety of bacterial antigens. Touart[25] made the following report on intracutaneous testing with autogenous vaccines:

a. Reactions are constant.

b. Early positive wheal reactions persist in spite of prolonged vaccine treatment.

c. The late positive reaction grows less or disappears in the course of vaccine treatment.

d. Attacks of asthma have followed the injections of vaccine which gave positive early or late reactions.

e. Old quiescent test or treatment sites have been lighted-up by subsequent injections of the same vaccine.

Of the 110 patients tested intracutaneously in this series, only 24 gave early positive wheal reactions and 16 showed delayed (24 to 48 hours) skin responses. The dosage was increased 0.05 cc. or 0.1 cc. at five day intervals, depending upon the reactivity of the subject. General and local reactions occurred in approximately half the patients treated. It was not unusual to produce an asthmatic attack by increasing the vaccine dose too rapidly. Typical allergic flare-up in the sinuses was witnessed on several occasions as a reaction to the injection of autogenous vaccines in very sensitive individuals. No serious reactions were encountered.

Benson[4] states that the reactions of intradermal tests in conjunction with the systemic effects of bacterial inoculation form the most reliable guide to vaccine therapy in cases of bacterial hypersensitivity of the respiratory tract. His requisites for success in vaccine therapy are the proper choice of foci for culturing, careful isolation of the predominant organism as determined by plate counts and standardization of dosage by cutaneous tests and systemic reaction. Skin responses to streptococcus hemolyticus when present in his patients were usually comparable with the ordinary 24 hours' response to the Dick test and streptococcus viridans and the nonhemolytic streptococci usually elicited cutaneous responses which are similar to those obtained with streptococcus hemolyticus. Kraft, Mothersill and Nestman[21] showed that about 80 per cent of their subjects gave immediate urticarial reactions to intradermal injections of bacterial antigens.

Schonwald and Deppe[22] obtained positive results in 84.4 per cent of 155 cases treated with vaccines made from airborne bacteria which are nonpathogenic. They have demonstrated, by skin tests and positive results of treatment, that an allergy to these bacteria exists in many cases. There were 60 cases of allergic rhinitis reported in this group.

A small group of patients in the present report had been treated previously with mixed stock vaccines without much relief of symptoms. They universally improved with autogenous vaccine therapy. The determination of which type of vaccine to use depends upon clinical experience rather than laboratory observations. Since antigenic qualities do not remain unchanged by artificial cultivation as evidenced by loss of capsules, diminution of virulence, etc., it is highly advisable that the organisms causing the infection be obtained and included in the vaccine as quickly as possible. The existence of serologically specific types of bacteria is well known, as in the case of staphylococcus aureus, which was by far the commonest organism encountered in the respiratory tract in this series and others,[3] hence the injection of one strain in a vaccine may not protect against another. The use of a properly prepared autogenous vaccine reduces to a minimum the risk of using a different strain and makes it preferable to a stock vaccine for therapeutic purposes. The ability to utilize freshly isolated strains of virulent bacteria unchanged by prolonged cultivation is the main advantage of an autogenous vaccine in addition to the fact that freshly prepared vaccines are probably more antigenic than old stock vaccines.[26]

In this study, 90 cases of hyperplastic sinusitis and asthma were either improved or markedly improved with this type of therapy. The treatment of hyperplastic sinusitis with autogenous vaccines, either obtained from cultures of the nose, sinus membranes at operation, sinus washings, bronchoscopic aspiration, nasopharynx or tonsils, has proved to be of great value, particularly in those patients whose atopic allergies were controlled but whose symptoms continued or those who lacked skin sensitivity to nonbacterial allergens. Goodale[27] believed that an allergic individual who shows negative skin tests is sensitive to bacteria. The findings in this study corroborate this view.

SUMMARY.

A study is presented of 110 patients with respiratory allergy who had bacterial infection as the predominant cause of their symptoms. They were from six and one-half months to 81 years of age and had symptoms from one month to 50 years.

They were all treated with autogenous vaccines prepared from infected foci; the predominant organism being the staphylococcus. Mixed vaccines of two or more bacteria were used in the majority of patients, and the average period of treatment was one year. Local and general reactions were not uncommon. Intradermal skin tests were positive in 40 cases.

Improvement or marked improvement was observed in 90 patients.

The data included in this study is additional evidence that bacterial sensitivity plays an important rôle in the pathogenesis of chronic rhinitis, sinusitis and asthmatic bronchitis.

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INNER EAR DEAFNESS OF SUDDEN ONSET.*†‡

JOHN R. LINDSAY, M.D., and JACOB J. ZUIDEMA, M.D.,
Chicago, Ill.

This report concerns those cases of inner ear deafness of sudden onset which cannot be attributed to such well known causes as suppurative labyrinthitis, tumors, inner ear trauma or known toxic involvement. These cases are also clinically differentiated from Ménière's disease or idiopathic hydrops of the labyrinth by the fact that they are characterized by one attack only, with permanent impairment of function, whereas in Ménière's disease there are recurring attacks of vertigo and a deafness and tinnitus which fluctuate in severity from time to time.

Factual information as to the pathological process underlying the loss of function in these cases of sudden onset has been lacking since the incidence is low and the condition is not fatal. Our impressions as to the probable pathology have been gained from the postmortem examination of cases in the

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*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 16, 1950.

†From the Division of Otolaryngology, University of Chicago.

‡Since this report was written a fourth case of bilateral profound deafness and loss of vestibular function, associated with nonluetic keratitis, has been observed. A brief history follows:

W. S., a 31-year-old male, was in good health until 1939 when he developed blurring of vision. One month later he became dizzy and began to vomit. Within a few hours he noted deafness on the right. A diagnosis of keratitis and VIIIth nerve deafness was made.

In October, 1941, he again developed an alternating keratitis, loss of balance and sudden deafness on the left. A tentative diagnosis of C.N.S. lues was made despite negative blood and spinal fluid serology, and fever therapy was instituted without improvement. Since that time he has had a constant roaring in his head.

In October, 1948, he had a recurrence of the keratitis which responded again to atropine therapy but the hearing loss has been permanent. He was first seen in our clinic on Feb. 4, 1950, at which time his ear, nose and throat examination was negative. Apart from profound bilateral nerve type deafness and absence of caloric responses to ice water, this appears to be a fourth case of Cogan's syndrome which differs from the other two severe cases only in that this patient has had head noises.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

various stages of labyrinthitis, and from such conditions as leucemia and Paget's disease of the temporal bone which have undergone an acute inner ear disturbance in the terminal stages. Some evidence as to the susceptibility of the sense organ to chemical or toxic factors has also been gained from animal experiments.[1]

An acute inner ear disturbance resulting in permanent damage to function is known to occur in the course of some diseases, chiefly those of an acute infectious nature, such as mumps, typhus, Ramsey-Hunt syndrome, etc., but those cases in which no infectious process could be demonstrated have usually been considered as due to vascular disturbance in the labyrinth.

Some idea of the incidence of sudden deafness and vertigo in which the pathologic process is doubtful is indicated by the fact that the cases to be presented were selected in approximately a three-year period, whereas the number of cases of definite Ménière's disease during the same period is estimated at three or more times that number.

For purposes of comparison and discussion the report includes four cases (Cases 1, 2, 3, and 4) as examples of more *common and well known types in which the acute inner ear* disturbance was associated with some known infectious process and, therefore, less rare. The 12 succeeding cases seem to fall into one group in which the etiology is uncertain.

The last three cases seem to fit into a specific syndrome in which the inner ear disturbance was associated with an ocular disease.

Case 1: W. W., 58-year-old industrial engineer, had a history of bilateral otitis media with suppuration for one week in 1914. His hearing was subjectively normal. Dec. 31, 1946, he developed malaise and a mild temperature elevation. After two days he developed an earache on the right, which was followed in 24 hours by a serous drainage. A few hours after the drainage began he developed a roaring tinnitus and vertigo with nausea and vomiting. The nausea and vomiting subsided in three to four days and the vertigo became progressively less severe but was still aggravated by quick movements when seen four months later. At no time did he have any headache.

Ear, nose and throat examination four months after the onset of disease was normal apart from bilateral exostoses of the external canals.

Audiogram showed profound nerve type deafness on the right (see Fig. 1).

Spontaneous nystagmus could be demonstrated after head shaking, but no postural vertigo could be elicited.

Caloric response to ice water was absent on the right and was hyperactive on the left.

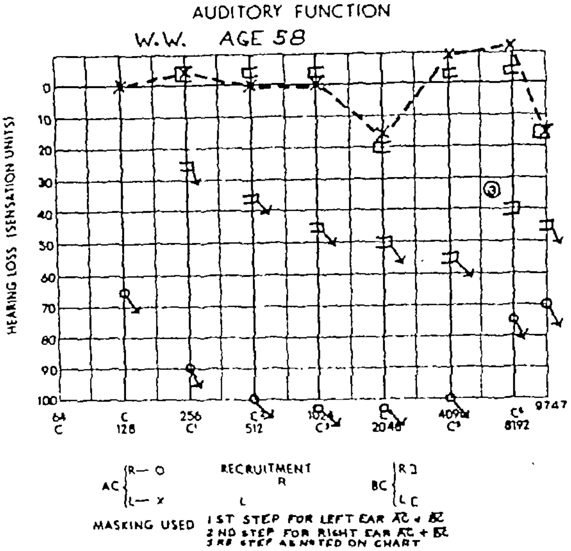


Fig. 1. Case 1. Acute suppurative otitis media followed in a few hours by tinnitus and profound deafness on the right with vertigo. The caloric response to ice water was absent on the right.

Comment: Although the history of frank suppuration from the ear was questionable, the diagnosis of diffuse labyrinthitis, serous or suppurative, seems definite.

Case 2: R. R., male, aged 27, was first seen on Jan. 26, 1939. At that time he gave a history of having had an acute osteomyelitis two years previously with marked temperature elevation, delirium, vertigo and hearing loss on the right. The ear did not discharge at this time. The vertigo subsided rapidly, but the deafness has been constant.

Examination of the nose and throat was normal. Both tympanic membranes were scarred but intact. An audiogram at the present time shows profound inner ear deafness on the right (see Fig. 2). The cold caloric response is normal in both ears and rotation tests are normal.

Impression: Metastatic embolus to the right inner ear with profound deafness and vestibular irritation.

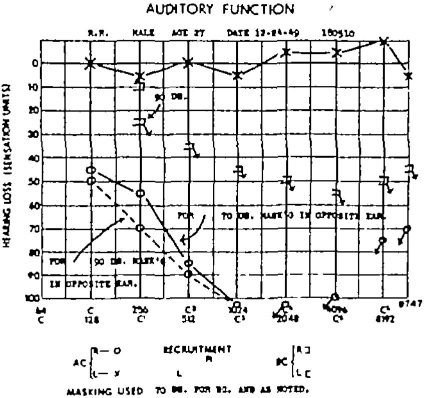


Fig. 2. Case 2. Sudden onset of vertigo and profound deafness on the right during acute osteomyelitis. Cold caloric responses were normal.

Comment: This case represents a sudden onset of deafness and vertigo without evidence of middle ear disease which occurred during an acute stage of osteomyelitis. The episode occurred before sulfonamide therapy was available. The preservation of normal vestibular function in the presence of profound deafness is a picture which is more suggestive of a toxic disturbance or of a metastatic embolus affecting the cochlear nerve than of a frank labyrinthitis.

Case 3: B. F., a 36-year-old housewife, gave a history of having had an eye infection and hearing loss, more severe on the right, 18 years before her visit on Dec. 18, 1946. Three months prior to this first visit, she had delivered a normal child, followed after one month by a sudden onset of head noises and almost complete bilateral deafness without vertigo. She had had bilateral chronic suppurative otitis media as a child.

Examination of the nose and throat were essentially normal. The tympanic membranes were intact but scarred. Audiogram revealed a marked bilateral nerve type deafness. Cold caloric responses were reduced bilaterally. The Kahn and Wassermann tests fluctuated between questionably positive and questionably negative. Antiluetic therapy was begun, but her hearing diminished still further while under treatment (see Fig. 3).

Impression: Late congenital lues. Marked bilateral nerve type deafness.

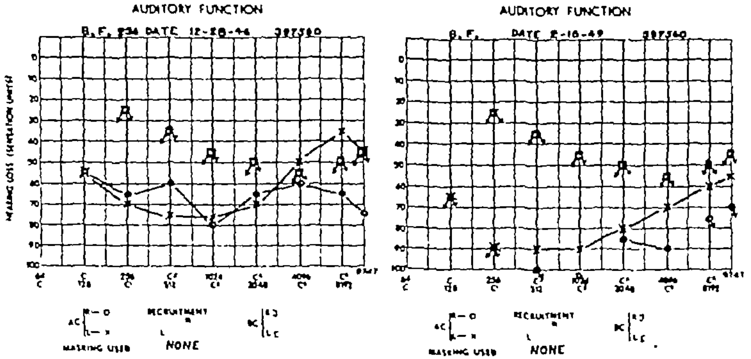


Fig. 3. Case 3. Sudden onset of severe deafness without vertigo one month after delivery of a normal child in a patient with congenital lues. The cold caloric responses were reduced bilaterally. The chart shows the progressive hearing loss during antiluetic treatment.

Comment: The history of sudden marked deafness three months before her first visit probably indicated a sudden exacerbation of deafness in her better hearing ear. The absence of vertigo at the time suggests a relatively mild exacerbation of the luetic process probably exerting a toxic effect on the auditory neural apparatus.

Case 4: E. L., a 20-year-old female student, has been under observation since childhood. She experienced an onset of sudden unilateral deafness while ill with epidemic parotitis when a child. There was no vestibular disturbance at any time.

Examination of the nose and throat was normal and both M.T.'s were normal in appearance. Auditory function was shown to be a profound deafness on the left, while the right was entirely normal (see Fig. 4). The cold caloric response was normal in both ears. No postural vertigo could be demonstrated.

Impression: Profound inner ear deafness, left, due to a toxic neuritis.

Comment: This case is included to illustrate the type of otitic complication which is frequently associated with mumps; unilateral profound and permanent deafness with little or no vertigo and no loss of vestibular function. This complication has occurred bilaterally in rare instances.

A toxic neuritis affecting the cochlear neural apparatus but sparing the vestibular sense organs is the usual interpretation but histopathological examinations have not been reported.

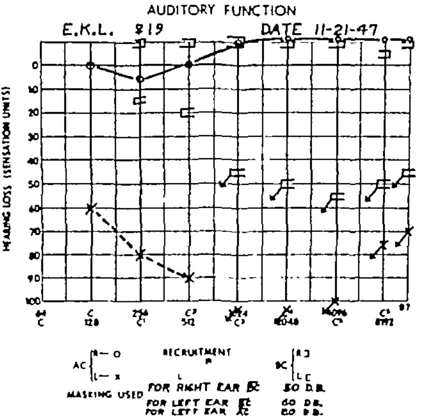


Fig. 4. Case 4. Audiogram of a patient who experienced sudden profound deafness without vertigo following mumps.

Case 5: A. N., a 28-year-old female, on Oct. 29, 1947, developed tinnitus and after a few hours deafness on the left. Twenty-four hours later she *developed vertigo with nausea and vomiting. She was in good health* at the time of onset but had had a severe cold one month previously. The patient never had had mumps but stated that at the time of her cold, her mother was recovering from a glandular swelling that was thought by her local physician to have been mumps. The patient was not seen until April 27, 1948. At this time she still complained of the deafness and a "sea-shell' tinnitus.

Examination of the nose and throat was essentially normal and the M.T.s were normal. Audiogram revealed the hearing to be normal on the right but profound nerve type deafness on the left (see Fig. 5). Cold caloric response was normal on the right and was slightly reduced on the left.

Impression: Profound inner ear deafness on the left of sudden onset, possibly due to subclinical mumps.

Comment: The ear complication in this case differs from that in Case 4 only in that vertigo was a prominent symptom at the time of the onset, and some loss of vestibular function has persisted.

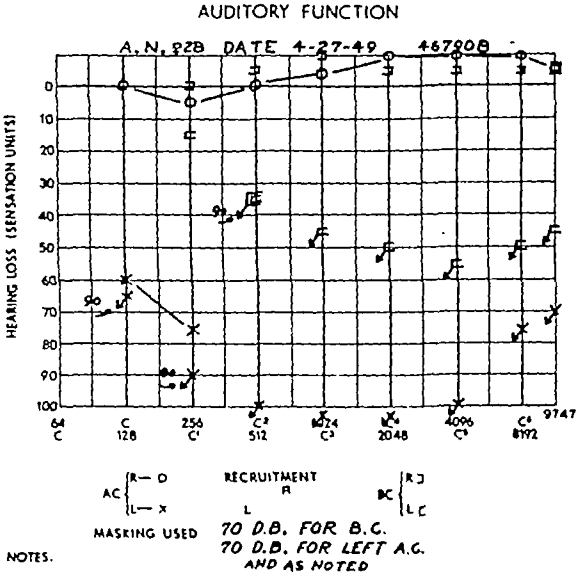


Fig. 5. Case 5. Sudden profound deafness and tinnitus on the left and vertigo without systemic disease one month after exposure to mumps.

The history of exposure to mumps one month previously suggests the possibility of a subclinical infection of this type with a complicating toxic neuritis of the VIIIth nerve.

Case 6: C. H., a 78-year-old female, was first seen March 5, 1949, with a history of bilateral decreased hearing for several years that was more pronounced on the right. History of previous suppuration in the right ear was given. Four weeks before her visit to the clinic she noted a low-pitched tinnitus on the left and a sudden marked hearing loss on that side.

Examination of the nose and throat was essentially normal. The right tympanic membrane was retracted and distorted, while the left was retracted. Hearing tests showed a moderate nerve type deafness on the right, and a profound nerve type deafness on the left (see Fig. 6). Cold caloric responses were normal on both sides. She gave a history of mild postural vertigo that was unchanged by the onset of sudden deafness on the left.

Impression: Moderate inner ear deafness on the right of long duration.

Profound inner ear deafness on the left of sudden onset. Etiology is doubtful.

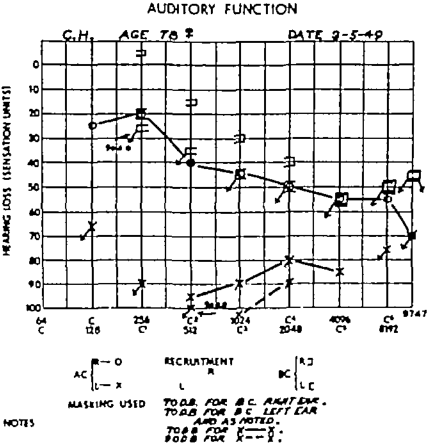


Fig. 6. Case 6. Sudden onset of tinnitus and profound deafness on the left without vertigo in a patient who had a previous bilateral deafness which was more severe on the right. The cold caloric responses were normal.

Comment: The sudden onset of profound unilateral deafness in the absence of any demonstrable infection at the age of 78 is suggestive of a vascular accident affecting the end-organ. Such an accident, in order to destroy hearing without interfering with vestibular function in any way, would necessarily be limited to the cochlear apparatus.

Case 7: M. Z. was a 54-year-old nurse. She was first seen April 15, 1938, stating that Jan. 15, 1938, she noted a click in her left ear which was followed by a persistent and severe hearing loss and a low-pitched tinnitus. She had no vertigo or diplacusis.

Nose and throat examination was essentially normal. Ear examination revealed normal M.T. bilaterally. Audiogram showed essentially normal function on the right but a profound inner ear deafness on the left (see Fig. 7). Cold caloric responses were normal.

Impression: Profound inner ear deafness, left, of sudden onset. Etiology is uncertain.

Comment: This case illustrates a sudden onset of unilateral profound deafness without vestibular disturbance. There was no history of any known infection at the time of onset.

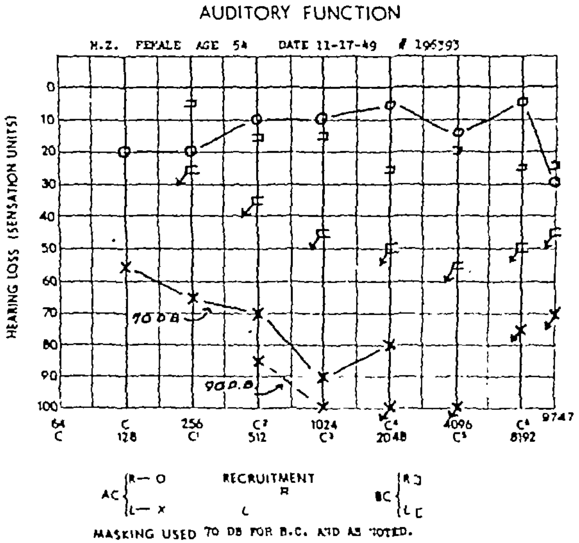


Fig. 7. Case 7. Sudden onset of severe deafness and tinnitus on left without vertigo. The cold caloric responses were normal.

The auditory complication was similar to that usually seen in mumps and is, therefore, suggestive of a toxic neuritis.

The age of the patient on the other hand might suggest a vascular accident. Such a vascular lesion would necessarily be localized to the cochlear apparatus.

Case 8: J. F. is a 36-year-old male who, on the night of June 23, 1949, while driving his car, had a feeling that something broke in his left ear and was followed by vertigo. He continued home and then vomited. He felt all right while lying down but vertigo returned on getting up. The following day he first noted deafness in the left ear. His vertigo had subsided in five days and at that time he was given injections of histamine and oral phenobarbital and nicotinic acid. Since his hearing did not improve, he presented himself at our clinic.

Complete neurological examination was normal apart from deafness on the left. Nose and throat examination was normal. The M.T. appeared normal. Audiogram revealed profound nerve type deafness on the left (see Fig. 8). Cold caloric response on the right was normal but there was no response to ice water on the left.

Impression: Profound deafness on the left with loss of vestibular function.

Comment: This case differs from the four previous cases in that the sudden onset of profound deafness was associated

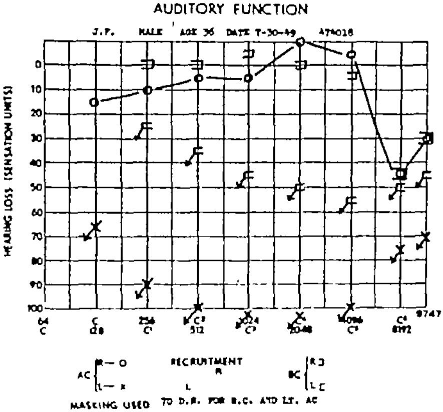


Fig. 8. Case 8. Sudden onset of profound deafness on the left and vertigo. The cold caloric response was absent to ice water on the left.

with a severe vestibular disturbance and permanent loss of vestibular function. There was no known infection at the time.

The diagnosis of a vascular accident in the labyrinth has been commonly made in such cases. The age of the patient, 36 years, is evidence against, but does not preclude, such a possibility.

Case 9: A. E. is a 40-year-old female who developed a sudden deafness with tinnitus in the right ear while sewing two weeks before her first visit on Aug. 28, 1949. That evening she developed vertigo with nausea and vomiting. The vertigo subsided in about three days. She had no known systemic infection at the time of the onset of symptoms. She also developed a blurring of vision at this time which persisted for two weeks. She had had intermittent drainage from the left ear since childhood.

Examination of the nose and throat was essentially normal. The right M.T. was normal, while the left was scarred but intact. Audiogram revealed a moderate nerve type deafness on the left and profound nerve type deafness on the right (see Fig. 9). Cold caloric response was normal on the left but was diminished on the right. There was no postural vertigo.

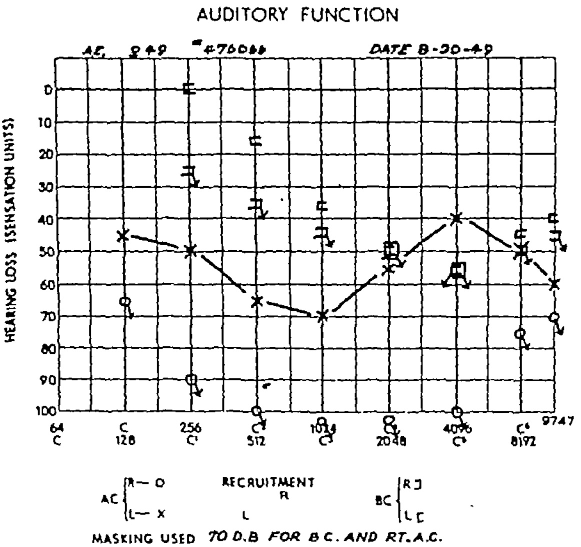


Fig. 9. Case 9. Sudden onset of profound deafness and tinnitus on the right and vertigo. The cold caloric response was diminished on the right. The left drum was scarred, due to previous suppuration.

Impression: Previous middle ear suppuration on the left with moderate nerve degeneration.

Profound deafness of sudden onset and moderate impairment of vestibular function on the right.

Comment: This case differs from Case 8 only in that vestibular function has been impaired rather than destroyed.

Case 10: M. F. was a 46-year-old female. She noted a sudden onset of deafness and roaring noises in the left ear in October, 1944, during a severe head cold. Noises began in the evening and the following morning she developed dizziness, nausea and vomiting which kept up for one week. The vertigo disappeared gradually but the deafness and tinnitus have persisted. There was no history of postural vertigo after the first attack had cleared, although a severe, sudden noise has caused momentary dizziness on two occasions since then. She had mumps at the age of 18, also measles, chicken pox and whooping cough.

Examination of the nose and throat was essentially negative and the M.T. appeared normal. Audiogram revealed normal hearing on the right and a moderate nerve type deafness on the left (see Fig. 10). Cold caloric responses were normal in both ears. No postural vertigo could be demonstrated.

Impression: Unilateral nerve type deafness of sudden onset with mild vestibular symptoms.

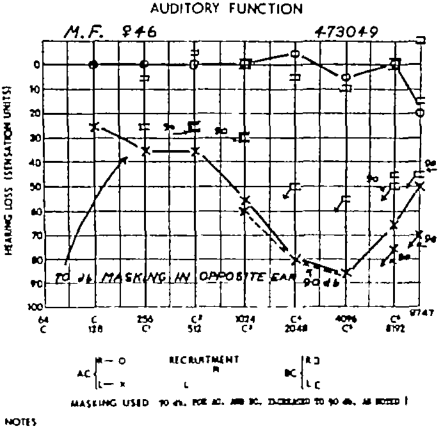


Fig. 10. Case 10. Sudden onset of vertigo and tinnitus and moderate deafness on the left during a severe head cold. The cold caloric responses were normal.

Comment: This case would appear to belong to the same group as Case 9 and to differ only in the matter of degree. Although a vascular accident in the inner ear is frequently assumed in such cases the fact that symptoms began during a severe cold raises the question of a toxic neuritis.

Case 11: S. N., a 29-year-old male, was first seen Dec. 2, 1949, and stated that 18 months before he had become dizzy while working in the sun. He was told that he had a "heat stroke" and was sent home. That evening he noted that he had a ringing in his left ear and that he could not hear the telephone with the left ear. The vertigo subsided quite rapidly, although he still describes what may be a mild postural vertigo. The hearing and tinnitus have not changed.

Examination of the nose and throat was essentially normal. The M.T. appeared normal to inspection. Audiogram showed the hearing to be normal on the right but a nerve type deafness on the left (see Fig. 11). Cold caloric responses were normal in both ears.

Impression: Nerve type deafness of rapid onset, left.

Etiology: Uncertain.

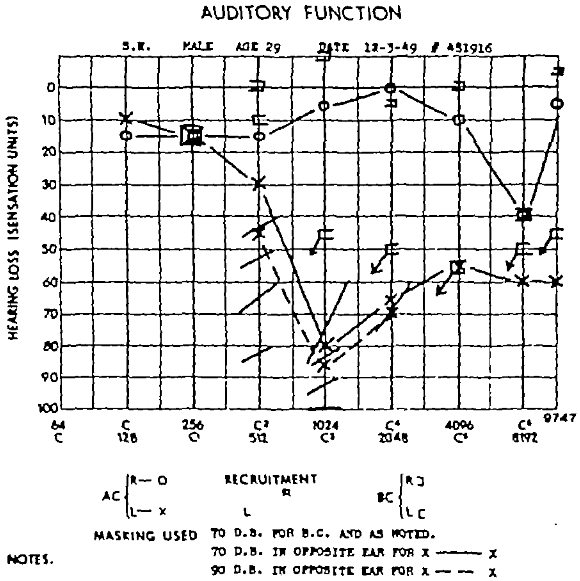


Fig. 11. Case 11. Sudden onset of vertigo followed by tinnitus and moderate deafness on the left while working in the sun. The patient still describes a postural vertigo but cannot be demonstrated. The cold caloric responses were normal.

Comment: This case apparently corresponds closely to Case 10 with the one exception, that the vertigo was complained of as a postural symptom for 18 months after the onset.

This patient was only 29 years of age and showed no evidence of systemic disease. A vascular accident such as a thrombosis or hemorrhage is, therefore, improbable.

Case 12: C. V. is a 27-year-old female who went to bed in the evening with a mild upper respiratory infection. On arising in the morning she had vertigo with nausea and vomiting. During the day when using the telephone she noted that she was unable to hear with the right ear. The vertigo passed away in about 24 hours, but for about one month she noted that she became dizzy very easily. She described no definite postural vertigo, but she believes that her hearing does fluctuate a little and that there had been some improvement.

On examination, Feb. 12, 1948, which was two weeks after the onset, the nose and throat examination was normal and the M.T. appeared to be normal. Audiogram revealed normal hearing on the left and a high tone nerve type deafness on the right (see Fig. 12). Cold caloric responses were normal on the left and reduced on the right. There was no postural vertigo demonstrated.

Impression: High tone nerve type deafness of rapid onset of uncertain etiology.

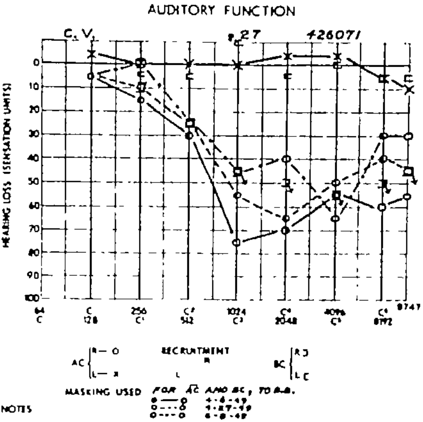


Fig. 12. Case 12. Vertigo and sudden moderate deafness on the right during a mild upper respiratory infection. The cold caloric response was reduced on the right. The chart shows the spontaneous hearing improvement in two months' time.

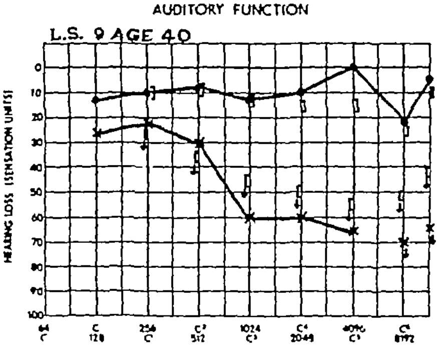
Comment: The onset of the unilateral deafness with vertigo was in this case associated with a mild upper respiratory infection. The severity of findings and the course of the disease corresponds closely, however, to other cases reported in this group and a similar etiology is, therefore, probable.

The audiogram illustrates the degree of recovery which occurred spontaneously.

Case 13 (previously reported in *Annals of Otology, Rhinology and Laryngology*, 56:3:541, Sept., 1947): L. S., female, aged 40, experienced a sudden onset on March 1, 1946, of screaming and whistling noises in the right ear, associated with deafness and a gradual onset of vertigo which reached a maximum about one week later. The vertigo has been postural, brought on especially by placing her head backwards, lying on one side, and after sitting up. It has not decreased in the period of one year since the onset. She is unable to tolerate high-pitched loud sounds or music. She has had no headaches. Her ears were normal previous to this episode. There has been no appreciable change in the symptoms during the past year.

The general examination, including the neurological, was essentially negative.

The nose and throat examination was negative. The tympanic membranes were normal. There was moderate high-tone nerve deafness in the right ear (see Fig. 13).



AIR COND. R — X
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BONE COND. R — [
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MASKING — BUZZER AT MAXIMUM INTENSITY

VESTIBULAR EXAM

NO SPONTANEOUS NYSTAGMUS IN ERECT POSITION

COLD CALORIC RESPONSES NORMAL BILATERAL

POSTURAL TESTS

(1) ON LEFT SIDE - VERTIGO AND NYST. ↺ 20 SECS.
(2) HEAD HANGING - VERTIGO AND NYST. ↻ 20 SECS.
(3) AFTER SITTING UP - VERT. AND NYST. ↻ 10 SECS.

OTHER POSITIONS NEGATIVE.

Fig. 13. Case 13. Sudden onset of tinnitus and impaired hearing in the right and gradual onset of vertigo. Postural vertigo was present one year after the onset.

No spontaneous nystagmus was present in the upright position. The fistula test was negative. There was a normal cold caloric response in each ear. The postural tests revealed a marked vertigo and nystagmus in the head hanging position, on the left side, and on sitting up. The positional nystagmus was counterclockwise in the left lateral position but clockwise in the head hanging position and after sitting up quickly.

Impression: High tone nerve type deafness, right, of uncertain etiology.

Case 14 (previously reported in the *Annals of Otology, Rhinology and Laryngology*, 56:3:541, Sept., 1947): H. R., female, aged 35, complained of deafness and noises in the left ear and dizziness. The symptoms came on suddenly three years earlier.

Vertigo was accompanied by vomiting at the onset. After about two weeks the dizziness had receded in severity and occurred only in certain positions. There were no further attacks, but the postural vertigo has persisted without further improvement until the present. It is noted particularly on lying on the left side. A buzzing noise in the affected ear and a sense of pressure or blockage varying in severity from time to time has been troublesome. Her hearing is greatly depressed.

There were no ear symptoms previous to the onset of the present complaints.

The general examination was essentially negative.

Local ear, nose and throat examination showed normal tympanic membranes. The right maxillary sinus was filled by a large polyp projecting into the nasal passage. There was no evidence of suppuration.

The auditory examination revealed profound nerve deafness on the left side but some residual hearing was present with adequate masking. There was normal hearing on the right (see Fig. 14).

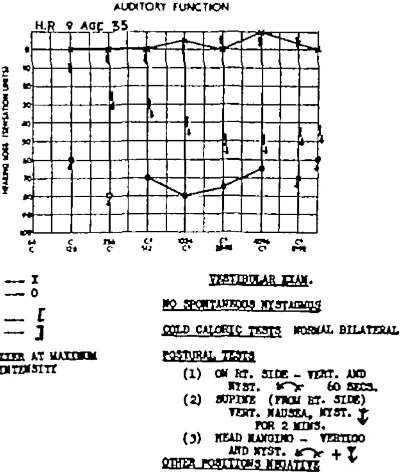

Fig. 14. Case 14. Sudden onset of tinnitus and severe deafness on the left and vertigo three years previous. Postural vertigo and unilateral tinnitus and deafness persist.

There was no spontaneous nystagmus in the upright position. The fistula test was negative. There was a normal cold caloric response in both ears. In the postural tests vertigo was accompanied by positional nystagmus in the supine and head hanging positions.

A radical antrum operation on the right revealed the antrum to be filled by polypoid and cystic tissue.

Nine months later the complaints referable to the left ear remained unaltered.

Impression: Profound deafness of sudden onset, left.

Etiology: Uncertain.

Comment: Cases 13 and 14 are very similar and can be discussed together. The possibility of a thrombosis due to an arteriosclerotic lesion affecting only the cochlear division of the arterial suply has been suggested to explain sudden deafness on the basis of vascular distribution to the labyrinth.

While a vascular accident might offer the best explanation for the apoplectic type of a Ménière's syndrome occurring as a single episode in a previously healthy ear after middle age, factual information regarding the pathology is at present inadequate. A toxic neuritis sometimes cannot be differentiated.

Case 15: H. M., female, aged 37, was first seen on Sept. 25, 1948, and at that time gave a history of severe right parietal headache one evening in May, 1945. She went to bed but was awakened by vertigo, nausea and vomiting. Three to four days later she developed a tinnitus and deafness in the right ear. She had no systemic disease at the time of onset. The vertigo subsided in a few days except when she assumed certain positions. This postural vertigo persisted for about three years and has gradually disappeared. After one year she believed the hearing began to return; but the tinnitus persisted, and loud sounds continued to cause an unpleasant sensation in the right ear. Her past history is contributory inasmuch as she was always susceptible to motion sickness and that she had migraine headaches since 1942, which seemed to have been controlled by histamine.

Examination of the nose and throat was normal, and the M.T. appeared normal. Audiogram revealed a high tone nerve deafness on the right (see Fig. 15), while hearing was normal on the left. The cold caloric response was normal on the left but was reduced on the right. No postural vertigo could be demonstrated on examination.

Impression: High tone nerve deafness and reduced vestibular function on the right of uncertain etiology.

Comment: This case demonstrates a vestibular disturbance associated with headache and unilateral deafness of rapid onset. The hearing loss and vestibular dysfunction were permanent. Postural vertigo persisted for about three years. The case corresponds closely to Cases 13 and 14, except for slight difference of degree.

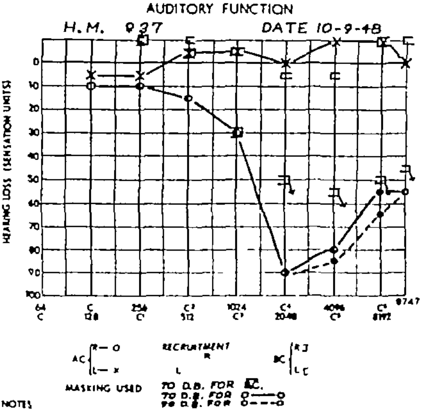

FIG. 15. Case 15. Sudden onset of tinnitus and deafness on the right, along with vertigo four years earlier. Postural vertigo persisted for three years, decreasing in the fourth year. The cold caloric response was moderately decreased on the right.

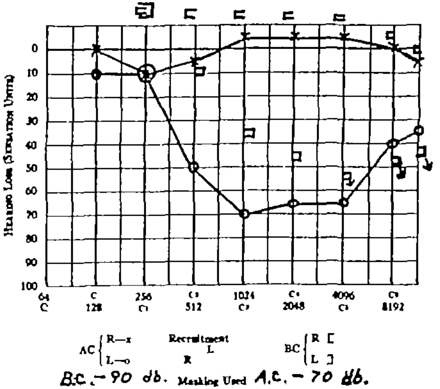

FIG. 16. Case 16. Rapid onset of tinnitus and moderate deafness on the left with vertigo. A postural vertigo persisted over one and one-half years. The cold caloric response was slightly reduced on the left.

Case 16: V. D., 24-year-old female, was in good health until July, 1946, at which time she developed a tinnitus that was followed in about two hours by vertigo with nausea and deafness on the left. The vertigo persisted about 10 days and required bed rest for the first two days, followed by postural vertigo which persisted until the time of examination over one and one-half years later. Positional nystagmus was demonstrated at the examination. Some spontaneous improvement in hearing was observed.

Examination of the nose and throat was essentially normal. The tympanic membranes were grossly normal. Audiogram was normal on the right, but there was a moderate nerve deafness on the left (see Fig. 16). The cold caloric response was normal on the right but was reduced slightly on the left. A postural nystagmus was present for 15 seconds only when the patient was lying on her left side.

Impression: High tone nerve deafness and reduced vestibular function on the left of uncertain etiology.

Comment: This case closely resembles Cases 13, 14 and 15 which had a sudden onset of inner ear deafness, tinnitus and vertigo. The vertigo subsided within a few days to become a postural symptom only, which was known to persist for as long as three years; however, this patient was only 24 years of age and was in good general health. A vascular accident in the inner ear could scarcely be considered as a likely etiologic factor. Although there was no history of any known infection at the time of onset of the episode the possibility of a toxic process affecting the inner ear must be considered.

DISCUSSION.

Sixteen cases of inner ear deafness of sudden onset have been presented, the last 12 of which must be considered as of indefinite etiology. The first four cases were presented only to illustrate types of sudden inner ear disturbance in which the etiologic relationship to infection was evident. These are all cases of permanent unilateral deafness of sudden onset with or without vestibular disturbance, occurring as a single attack. The localization of the lesion to the inner ear seems definite in view of the association of vestibular symptoms with moderate to profound unilateral deafness.

The sudden onset, the single attack only, and the permanent loss of function are sufficient to differentiate these from Ménière's disease (idiopathic hydrops of the labyrinth). In

Ménière's disease there are remissions and exacerbations of tinnitus and low-tone deafness, and usually progression of the deafness over a period of years. Although there are repeated attacks of vertigo, vestibular function as shown by the cold caloric tests is usually normal in the early stages. Because of these facts it seems improbable that the same pathologic disturbances occur in both diseases or that they are due to the same etiologic factor.

Deafness of sudden onset, sometimes associated with vertigo, is known clinically to occur on a toxic basis as a complication of some infectious diseases. These are mainly virus in type. Involvement of the vestibular apparatus has not usually been proportionate to the auditory disturbance and may even be absent. There is, however, no constant pattern as regards proportion of hearing loss to vestibular dysfunction as a result of a toxic process.

Vascular disease of various types is also considered a cause for deafness of sudden onset with or without vestibular disturbance.

Hemorrhage is perhaps considered first when vascular disease is thought to be the etiologic factor. Several of the patients reported here were still in their twenties and in apparent good health; therefore, hemorrhage is an improbable explanation. None was known to have had blood dyscrasias, and none had been exposed to head trauma.

Patients who had developed vestibuloauditory symptoms in *association with leucemia*[3] *and whose temporal bones had* been subsequently examined were found in some instances to have a serofibrinous exudate in the labyrinth and in some to have fibrosis and a labyrinthitis ossificans. These findings in cases of leucemia appear to have been the main basis for the diagnosis of hemorrhage in the labyrinth in cases such as those presented in this report.

Thrombosis, occlusion or spasm with resulting ischemia are other vascular conditions that are not easy to differentiate. The vascular supply of the cochlea is such that a thrombus might affect the entire auditory sense organ and theoretically might account for deafness without vertigo. The majority of cases presented here, however, have shown varying degrees of involvement of both auditory and vestibular function and, therefore, are not explainable on the basis of vascular occlusion; also, the occurrence of the disease before 30 years of age in a significant proportion of cases is evidence against vascular accident.

A vasomotor disturbance as postulated by Brunner[3] must also be considered. The pathologic findings in a case of Paget's disease of the temporal bone which had experienced a severe vestibuloauditory disturbance shortly before death were described by him as otitis interna vasomotoria. While there seems to be little evidence to indicate that the type of pathologic change demonstrated in advanced Paget's disease might occur in otherwise normal young adults, it must be admitted that a vasomotor disturbance offers a possible explanation.

In searching for the etiologic factor of Ménière's disease, allergy has been suggested and has received much consideration. Rather infrequently has there been direct evidence to associate atopic allergy with Ménière's disease. Intrinsic or physical allergy has been postulated as an etiologic explanation. Inasmuch as these cases are characterized by a single episode in a lifetime, an allergic factor appears unlikely.

THE SYNDROME OF DEAFNESS AND VERTIGO AND NONLUETIC INTERSTITIAL KERATITIS AND IRITIS.

Case 17: H. N., 38-year-old female, noted reddening of her eyes in November, 1943, which subsided over a period of three months. She had a slight but daily temperature elevation for one month beginning in December, 1943. About the time that her eyes improved she began to have tinnitus on the left, which soon became bilateral and was associated with bilateral deafness and vertigo on movement.

She was first seen March 14, 1944, at which time the general physical examination was essentially normal. There were no abnormal findings in the nose and throat and the M.T. appeared normal. Neurological examination was also normal apart from the vestibuloauditory findings. Audiogram revealed a bilateral nerve type deafness (see Fig. 17). Cold caloric responses were hypoactive in both ears. The eye disease was

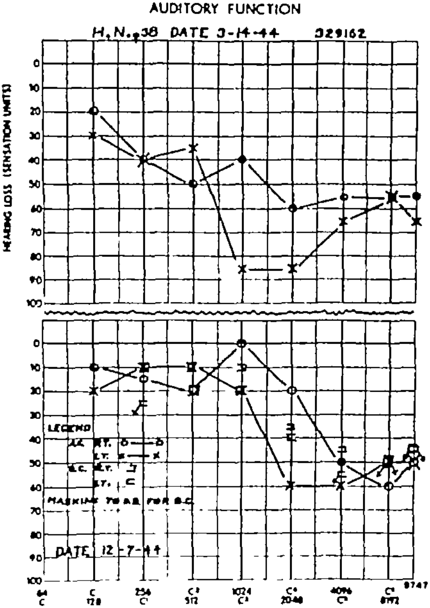

Fig. 17. Case 17. Bilateral nerve type deafness and tinnitus of rapid onset with vertigo and reduced cold caloric responses associated with bilateral iritis. The spontaneous recovery is illustrated.

diagnosed as bilateral iritis with secondary glaucoma on the left. She had been followed since that time and there has been some recovery of hearing.

Impression: 1. Bilateral nerve type deafness with reduced caloric responses. 2. Bilateral iritis with secondary glaucoma on the left.

Case 18: J. M., male, aged 37, was well until the morning of Oct. 20, 1948, when on arising he noted nausea, vomiting and vertigo. Twenty-four hours later he noted bilateral tinnitus and deafness which was progressive for 24 hours. The vertigo subsided in three days and by the end of one week the hearing was normal on the right and but slightly impaired on the left. He was well until Feb. 1, 1949, when he again noted vertigo with nausea and vomiting for a few hours. A lumbar puncture done at that time was reported to be normal. Five days later he developed pain and "conjunctivitis" in the left eye which lasted three days, but one week later was noted in the right eye. After that time the complaint alternated from one eye to the other. Feb. 25, 1949, he began to develop a blurring of vision between attacks of pain and a profound deafness developed. Skull X-rays, EEG, blood, urine and complete neurological examination were reported to be normal. He was then placed on daily histamine and vitamins without change.

He was first seen in our clinic April 6, 1949, at which time the general physical, neurological and nose and throat examination were normal. The tympanic membranes appeared normal. The audiogram showed a profound bilateral deafness (see Fig. 18). Caloric response to ice water was absent in both ears. There was no postural vertigo. Examination of the eye was reported as showing a nonluetic interstitial keratitis.

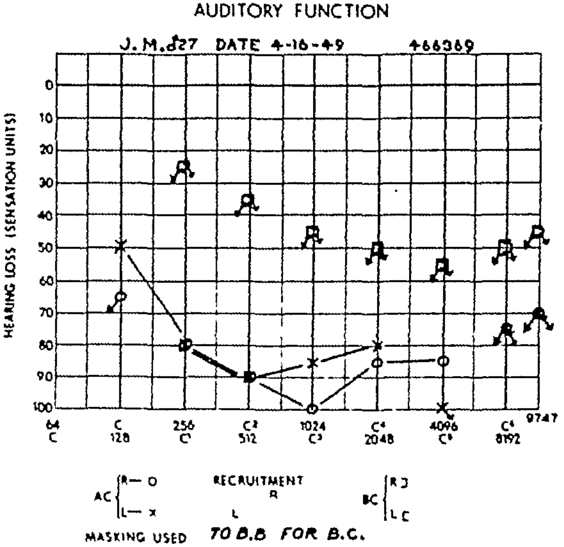

Fig. 18. Case 18. Rapid onset of bilateral deafness and vertigo associated with a bilateral nonluetic interstitial keratitis. The cold caloric responses to ice water were absent.

Impression: Nonluetic interstitial keratitis associated with profound bilateral inner ear deafness and loss of vestibular function.

Case 19: F. Di P., male, 26 years of age, developed in September, 1946, a sudden "conjunctivitis" with a right subconjunctival hemorrhage. The eye pain cleared in two days apart from a blurring of vision but was followed by bilateral tinnitus and 12 hours later by vertigo with nausea and vomiting. Three days later there was a rapid onset of profound deafness on the right and a severe loss of hearing on the left. He was treated with sulfonamides and penicillin for three and one-half weeks, followed by streptomycin (period unknown), after which the residual hearing on the left was lost. In March, 1947, he developed cardiac symptoms, palpitation and chest pain. The condition was controlled, but in November, 1948, he developed hemoptyses which subsided when the cardiac condition was again controlled.

When first seen in our clinic, Feb. 10, 1949, his general physical examination revealed an enlarged but compensated heart. Neurological examination was essentially negative. Eye consultants diagnosed a bilateral nonluetic interstitial keratitis. The nose and throat examination was essentially normal and the M.T. were normal. Audiogram showed a profound bilateral inner ear deafness (see Fig. 19). Cold caloric response was absent to ice water in both ears. There was no postural vertigo.

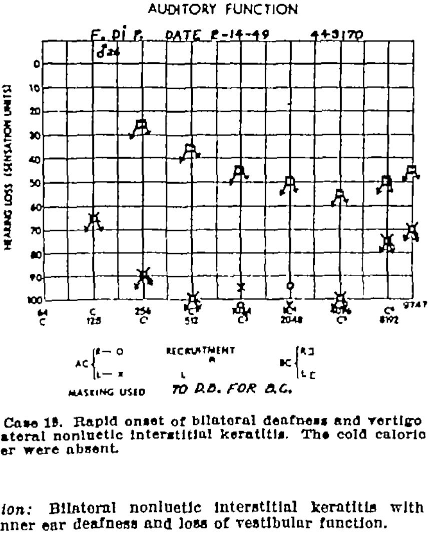

Fig. 19. Case 19. Rapid onset of bilateral deafness and vertigo associated with a bilateral nonluetic interstitial keratitis. The cold caloric responses to ice water were absent.

Impression: Bilateral nonluetic interstitial keratitis with profound bilateral inner ear deafness and loss of vestibular function.

Comment: Cases 17, 18 and 19 are all very similar and apparently belong to a specific syndrome. They differ from the previous cases in that the inner ear dysfunction is bilateral and associated with eye complications. Two of the cases had a single attack while the third had two attacks. Only one case had partial recovery of both ocular and inner ear function.

This syndrome differs from the syndrome of uveoparotid fever, which is usually characterized by a chronic painless parotitis, uveitis, facial paralysis and other neurologic changes in about 50 per cent of the cases.[4] The neurologic changes are mostly peripheral in type and are usually unilateral. Dysphagia is common and a nerve deafness is occasionally seen. The syndrome is frequently seen in patients with Boeck's sarcoid and the presence of lesions, on biopsy of the parotid, which resemble noncaseating tuberculosis has led many to believe that uveoparotid fever is a complication of Boeck's sarcoid.[5] The cases reported here show only an interstitial keratitis or iritis and inner ear dysfunction of sudden onset without neurologic manifestations. These findings do not correspond to those of uveoparotid fever and suggest a different syndrome. Cogan reported a series of four cases and cited a fifth which had been previously reported in which there was a nonluetic interstitial keratitis and inner ear dysfunction.[6] The order of appearance of symptoms was variable and the duration of progression was up to several weeks. Only one of the five cases demonstrated improvement and only after a period of years. In our cases as well as those of Cogan the diagnosis of lues appeared to be excluded. One case received aureomycin therapy without improvement, but this was over two months after the onset. It is apparent that our three cases as well as those of Cogan belong to the same syndrome. Although the etiology is uncertain, the inflammatory nature of the disease seems definite. Some type of virus disease seems most probable.

SUMMARY.

Sixteen cases have been presented of unilateral inner ear deafness of sudden onset with or without vestibular symptoms. The first four cases represent types in which the ear complication was associated with a systemic disease. Case 1 was apparently a diffuse labyrinthitis secondary to middle ear infection. Case 2 was probably a metastatic involvement of the inner ear during acute osteomyelitis. Case 3 was toxic in nature due to an exacerbation of a congenital luetic process in the ear. Case 4 represents the toxic neuritis of the audi-

tory neural apparatus associated with epidemic parotitis. The next 12 cases of unilateral sudden inner ear deafness with varying degrees of vestibular disturbance are of uncertain etiology. It cannot be definitely assumed that all 12 cases have the same etiology, but the similarity of this clinical picture strongly suggests that they are of the same type. A vascular accident in the labyrinth has commonly been the explanation in cases such as these. The high incidence in normal adults below 30 years of age argues against such a vascular accident. A vasomotor disturbance affecting the inner ear has been suggested and although hypothetical must be given consideration. Toxic involvement of the inner ear is a well known occurrence in infections of various types, some of which have been illustrated. In these cases of toxic neuritis, no common pattern exists as to proportionate vestibular and auditory dysfunction.

A toxic neuritis would provide a favorable explanation for the 12 cases presented of indefinite etiology and must be seriously entertained. The onset following an acute upper respiratory infection in two cases may give some support to this explanation.

The last three cases demonstrate a specific syndrome of bilateral inner ear dysfunction associated with an inflammatory ocular disturbance. Two of these had profound loss of auditory and vestibular function while the third had a less severe disturbance followed by partial recovery of function. The inner ear disturbance in this syndrome can be assumed to be toxic in nature, associated with an inflammatory process of unknown etiology.

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SURGICAL CORRECTION OF ANTERIOR WEB OF THE LARYNX.*†

ROBERT C. MCNAUGHT, M.D.,
San Francisco, Calif.

Anterior web of the larynx has always been a difficult problem. Until about 1880, when Schroetter,[20] of Vienna, started using indwelling cores of hard rubber and tin, the surgical treatment of stenosis of the larynx was considered hopeless. As recently as 1942, Loch and McHugh[13] state that surgery of anterior webs should never be attempted for improvement of voice only. They surveyed the literature to that time and were apparently impressed by the high degree of failure with any of the variations of the pioneer methods. These were three: repeated bouginage, splitting of the larynx by external operation with excision of scar, usually with the placement of a skin graft held by a "stent" of some sort, or the prolonged wearing of an O'Dwyer[16] tube or some modification of it. The methods were often combined.

Delsaux,[6] Schmiegelow,[19] Negus[15] and later Arbuckle,[1] Lynch,[12] New,[8] Figi[9] and others write of external methods, certainly inevitable in those cases of extensive trauma or obliteration of the lumen. Negus gives a good review of the problem to 1938. Hard and soft rubber tubes and plugs, metals including tantalum, and most recently acrylic forms have been employed, with and without skin grafts. For congenital or other webs of the thinner variety, Jackson, Clerf,[5] Tucker[21] and others advocate repeated bouginage sometimes after incision along one cord edge or in the midline with special knives

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif, Jan. 22, 1950.

†From the Division of Otolaryngology, Stanford University Medical School.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 10, 1950.

or radio knife. Triangular bougies and heated bougies have been used. Jackson urges conservatism in children, for use of the larynx stimulates its growth.

Haslinger,[10] using his directoscope, placed a silver plate between the severed cords and anchored it there with a wire

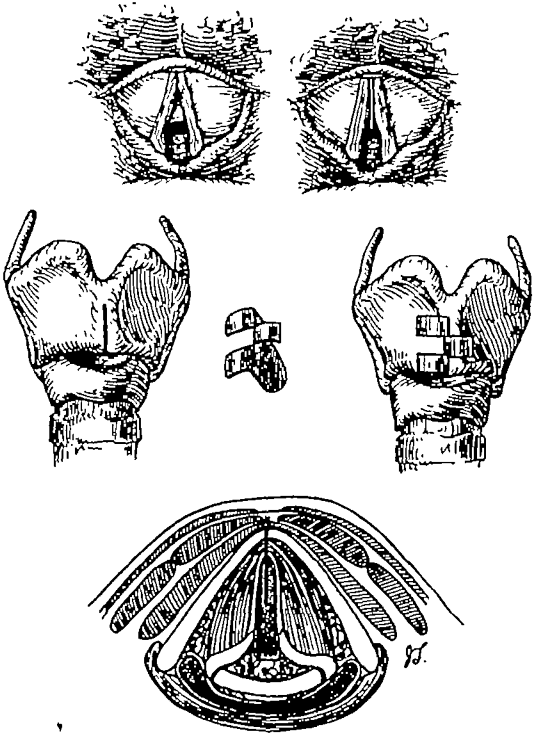

Fig. 1. Lower figure is a horizontal section at the level of vocal processes of arytenoids. It is schematic, since both right and left tabs would not show in one section. Note that the thyrohyoid muscles are sewed together in the midline to prevent outward displacement of the plate.

through the thyroid cartilage at the anterior commissure fastened to a button on the skin. In one case the plate cut through the thyroid cartilage and had to be removed by external incision. In another of the patients, carcinoma of the larynx was diagnosed six years later. Chondritis or the fear of it limited the use of this promising method. It was revived in 1948 by Poe and Seager,[17] although their modification of having the wire hug the inferior edge of the thyroid rather than to go through it would seem to permit the plate to be pushed backward by healing in the angle. Haslinger's method had the fundamental defect of all schemes that disregard the relative motion between the larynx and the skin and subcutaneous tissue. The earlier workers certainly did not lack ingenuity. We can profit by their experience with the added advantage of modern chemotherapy to minimize complications.

In my opinion, the real solution of the anterior web problem was envisioned by Iglauer.[11] In 1935, in an article which is an excellent review of the subject, he pointed out the similarity to the problem of syndactyly and reasoned that if a tract could be established first at the extreme angle, the cords would stay apart when later separated. All gaping incisions heal by fibrin strands bridging across the angle and contracting to literally sew the edges together, advancing the angle as this is accomplished. The place to check this undesired healing is at the apex. The mechanical problem of congenitally fused fingers defeated surgeons until it was realized that the important area was at the apex. When an epithelialized tract was established at this point by allowing a seton-like suture to remain in place a long time (as in the piercing of an earlobe), a subsequent incision to the free edge would remain open despite apposing raw surfaces.

Iglauer inserted the ring from a watch chain through the anterior commissure in one case. It was the type of ring in which a segment can be retracted against a spring. Due to technical difficulties the case was not entirely successful but the principle appealed to me as being the correct one; how-

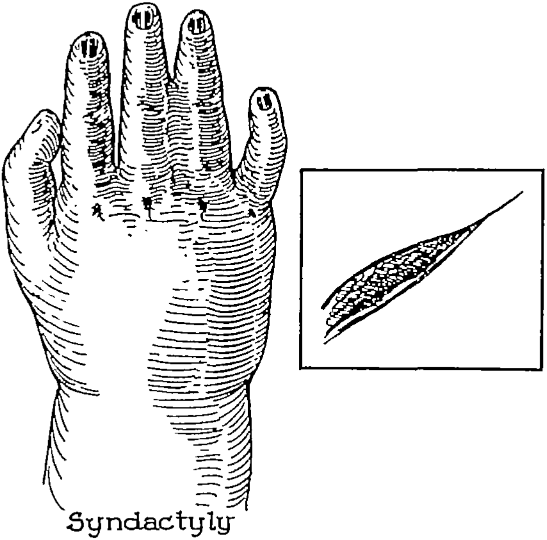

Fig. 2. Inset shows typical wound healing with fibrin strands bridging across the angle. Their contraction pulls the edges together, advancing the angle and literally sewing the wound closed.

ever, in the cases I have seen, no matter how deceptively thin the crescentic free edge of the web appeared, the webs were all too thick at the base for a small ring. A safety pin, closed subglottically, would be better, but I have not yet had a chance to try it.

In our experience congenital web of the larynx is not a problem commonly met. Stenosis from scar tissue web seems to have been more frequent when typhoid and lues were not so well controlled and when intubation of the larynx was freely employed. In the early days of my practice I inherited two adolescents who had undergone years of laryngeal dilatation by bouginage in an effort to get rid of tracheal cannulae. Both were the result of dense scarring from improper intubation. I succeeded in one case but the other patient gave up. Another patient at about the same period had a luetic stricture, finally treated by the method of Schmiegelow; that is, fissure with excision of scar and placement of a rubber tube secured by a silver wire transfixing the tube and both thyroid alae. The tube was removed by direct laryngoscopy three months later; but a long period of dilatation with the triangular bougies of Clerf proved necessary, because the cricoid had been involved in the chondritis.

These experiences made me reluctant to employ laryngeal bouginage or other methods of fighting scar tissue if any better way could be found of accomplishing the purpose. It was at about this time that Iglauer's paper appeared.

The case to be described is not one of a thin web but rather of stenosis resulting from dense scar tissue occupying the anterior one-third of the larynx. It extended subglottically a full centimeter below the level of the cords, fusing them together. The method I employed developed from an effort to apply the principle described, that is, to interfere with healing at the apex. Others as noted above had attempted this, but not all have stressed the importance of the apex.

REPORT OF CASE.

Mrs. B. was first seen 18 years ago when in her late thirties. She was, and still is, a saleswoman in a department store. She complained of loss of voice of three months' duration. She had a sessile papillary growth exactly in the anterior commissure, the attachment extending subglottically. This was removed by indirect laryngoscopy by my associate in September, 1931, and in the succeeding weeks several additional bits of tissue were taken for biopsy. There were some irregular nodules on each cord and the anterior one-third of the left cord showed a pale subglottic swelling. Some sections showed giant cells and epitheliod cells which Dr. Ophuls considered suspicious of lupus or tuberculosis. Voice rest and quartz light treatment to the larynx were employed. Some months later the anterior commissure was filled with granulations forming a web. This was punched out in August, 1932, and February, 1933. The probability of tuberculosis seemed more and more remote as the patient's general health remained good. She had some old scars at the apices, but repeated sputum examinations were negative. The Wasserman was negative.

She had returned to work although the voice was very poor. Through the intervening years she has been seen from time to time for other things, the web gradually growing thicker and denser. Not until 1948 was the patient desirous of having anything further done to the larynx.

By this time she realized that she was getting very tired from the continual effort of talking, required in her work. Dyspnea was beginning to be evident. The anterior one-third to two-fifths of the larynx was filled in by dense scar tissue.

In December, 1948, the following operation was devised and carried out under local anesthesia: a transverse incision was made through skin and platysma in a skin fold at mid thyroid-cartilage level. The ribbon muscles were separated in the midline down to perichondrium, which was incised in the midline but not otherwise disturbed. A thin bladed circular saw about 2 cm. in diameter was used to make a midline slot in the lower two-thirds of the cartilage only. It was a saw intended for the "Handee" tool but the mandrel fits the usual dental handpiece. The slot was made from the cricothyroid membrane upwards but stopped short of the thyroid notch, so as to leave a bar of cartilage connecting the two alae. The purpose was to prevent any overriding or displacement which might have occurred if the separation had been complete.

Fig. 3.

While an associate was observing the interior via direct laryngoscopy, a thin bladed sharp cataract knife was inserted through the cricothyroid membrane and brought up into the slot so as to divide the scar tissue in the midline. Care was taken not to injure the posterior wall and to bring the incision high enough to free the cord edges.

A plate of tantalum, 0.007 inches thick, was then placed through the saw cut in the cartilage like a center-board keel. It was trimmed so as to extend about half way back in the midline of the larynx, far enough to keep the raw surfaces apart but not so far as to interfere with the arytenoids. It reached from the cricothyroid membrane level to just above the upper surfaces of the cords. Posterior displacement was prevented by turning down tabs over each thyroid ala outside the perichondrium. The ribbon muscles were brought together in the midline over these tabs so as to prevent the plate from working outward. The fascia and skin were then closed without drains. Penicillin was given.

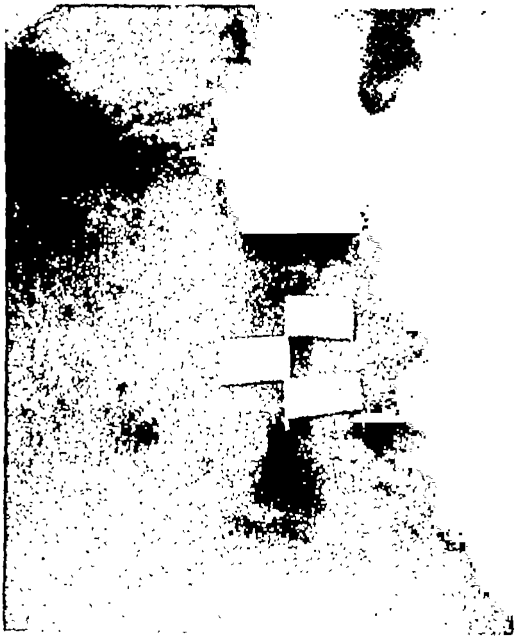

Fig. 4.

There was very little reaction. The patient swallowed normally the next day and left the hospital on the third day. For three days she inhaled penicillin dust t.i.d. and about this time was permitted to talk, since cough had not resulted in any subcutaneous emphysema. Her voice was better with the plate in than it had been before and breathing was very free. Fluoroscopy and films showed the plate to move with the larynx, not subject to external forces.

She returned to work. Frequent observation thereafter showed no inflammatory reaction to the presence of the tantalum at any time and it was judged after two months that epithelization was complete. She re-entered the hospital for removal of the plate. The incision was reopened and the plate removed without difficulty. There was no evident reaction outside the larynx. At this second hospital entry an attempt was made to take pictures of the plate *in situ* by direct laryngoscopy.

A recording was made of the patient's voice before the procedure was attempted, then during the time the tantalum plate was in place, and finally one 10 months after its removal.

The patient had promised to visit Dr. Paul Moses for voice training but so far has not done so. Abnormal habits of voice production had been necessary through the years of stenosis, and these have carried over to some extent. The larynx now looks so normal that I am sure better function is possible.

SUMMARY.

The problem of scar tissue web of the anterior third of the larynx is approached by applying the surgical principle of preventing healing from starting at the apex.

A case is described wherein a thin plate of tantalum was placed in the midsagittal plane of the larynx like a centerboard keel, through a slot in the thyroid cartilage made via external incision. It caused little reaction and was left two months. Epithelization was complete at that time and the plate was removed by reopening the transverse neck incision.

A recording was made before, during and after the pressure, demonstrating improvement in voice.

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Note: These references comprise a mere sampling of the literature on the subject. Many of the more important articles are not mentioned. McHugh and Loch[13] cite 167 references to 1942.

THE LIMITS OF IMPROVEMENT OF HEARING FOLLOWING THE FENESTRATION OPERATION.*

H. DAVIS, M.D., and T. E. WALSH, M.D.,
St. Louis, Mo.

INTRODUCTION

In the fenestration operation for clinical otosclerosis a new pathway for the entrance of sound to the inner ear is established to take the place of the pathway through ossicles and oval window. The oval window has been closed more or less rigidly by the otosclerosis. As a part of the operation the ossicular chain is broken and the head of the malleus and the incus are removed. The drum membrane remains, covering the round window in a much-reduced middle ear. Sound reaches the fenestra (and round window) without benefit of the normal impedance-matching device of drum, ossicles and oval window. The theoretical (maximum) gain from such matching in transmitting sound from air to the fluid of the inner ear is about 30 db. Animal experiments[1] indicate a rise in threshold of about 28 db when the ossicular chain and the eardrum are removed (in the cat). *It is reasonable to expect, therefore, that after the best possible fenestration there will still be a loss of sensitivity of at least 20 db compared to normal.* The minimum residual loss may be less than 30 db if the normal benefit from drum and ossicles is less than the theoretical maximum of 30 db. The residual loss will be greater if the fenestra is not fully mobile, if there are cancellation effects between round window and oval window, or if the sensitivity of the inner ear is reduced for any reason.

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*This work was begun under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under Contract V1001M-577 between Veterans Administration and Central Institute for the Deaf. The routine testing and the collection of data were transferred to the Hofheimer Audiology Laboratory at McMillan Hospital. The work there is supported by a grant from the Hofheimer Foundation.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 20, 1950.

Several additional self-evident propositions are relevant to any test of our major hypothesis.

1) The standard error of measurement of the test for auditory acuity will contribute to the scatter of the results. The scatter introduced thereby will be in the shape of a normal distribution curve. [For Auditory Test No. 9 (recorded spondee words) the standard error of measurement obtained from tests on 55 patients in the present series is 3.1 db.][2]

2) "Normal" inner ears are not identical in sensitivity but will undoubtedly vary in random fashion within a few decibels of the average.

3) The acoustic efficiency of post-operative fenestras will also vary about a mean. This distribution will not necessarily be "normal" but may have a "tail" of relatively inefficient fenestras.

4) The sensitivity of the inner ears in patients with clinical otosclerosis will vary in the direction of impairment of hearing (nerve deafness) by more than the normal amount. High-tone nerve deafness is known to be present in the numerous cases that are clinically classified as "mixed deafness." In many of these cases a discrimination loss is demonstrated by the "PB" word lists.[3] The threshold for speech, as measured by Test No. 9, may be elevated by such nerve deafness. In fact, a nerve deafness involving frequencies 512 and 1024 need not necessarily cause a discrimination loss, providing the loss for the higher speech frequencies is not severe. Any such "nerve component" in the hearing loss for speech will obviously not be eliminated by the fenestration operation and will remain as an addition to the unavoidable conductive loss of 20 db due to absence of ossicles. The sum of these various factors will determine the minimum residual (post-operative) hearing loss for any particular case.

5) There is a minimum amount of conductive hearing loss that can possibly be produced by even complete

fixation of the stapes. Sound will be conducted to the inner ear by bone conduction and/or via the round window even if all movement of the stapes is abolished. From general acoustic experience in sound exclusion we may expect that the threshold for such alternative pathways of hearing will lie about 60 db above normal; *i.e.* at about the level of the phantom audiogram produced by an unmasked normal ear opposite a totally deaf ear under test. (Such a 60 db loss means that only one millionth of the normal amount of acoustic power reaches the inner ear.)

Our original hypotheses did not include the effect of fixation of the stapes on the bone-conduction threshold. This effect has been demonstrated recently by Carhart.[4] It is a conductive loss. Due to the increase in stiffness of the acoustic system in the *inner* ear when the oval window is rigidly closed by an immobile stapes, most sound waves are less effective in producing movement of endolymph regardless of how the sound waves are conducted to the inner ear. The "Carhart effect" appears as an elevation of the threshold for bone conduction, amounting to a maximum of 15 db at 2000 cps, but it is *not* due to nerve deafness. It is one of the factors that determines the exact value of the maximum conductive hearing loss that can be measured by air conduction. It is eliminated by the fenestration operation. If the residual cochlear function or the amount of "nerve impairment" is assessed by bone conduction, allowance must be made for this factor. As will be seen below we actually did allow for it in interpreting our own bone-conduction measurements, without specifically recognizing its existence, when we established our norms for bone conduction by averaging the *pre*-operative bone-conduction thresholds of the ears that showed the lowest *post*-operative thresholds by air conduction.

A corollary of our general hypothesis is that any hearing loss more than the maximum conductive hearing loss by air (not yet closely defined, but about 60 db) must include *1)* some nerve deafness and/or *2)* some *additional unusual* form of sound exclusion such as might be produced by rigid closure of the round window. Neither of these two conditions will be alleviated or circumvented by fenestration; therefore, the *greatest possible gain for speech* that can be expected from fenestration is (maximum conductive loss) – (minimum unavoidable residual loss). Substituting the numerical values given above we have

$$\text{Maximum possible gain} = 60 - 20 = 40 \text{ db}$$

TESTS OF HEARING

We have tested our hypotheses by examining the results of 196 consecutive cases of fenestration performed by one of us (TEW) at McMillan Hospital (Washington University).

In none of the operations in question was a cartilage stopple employed, but nearly all of the cases operated by TEW without stopple (other than revisions) are included. No revisions of previous operations are included. The series begins arbitrarily in September, 1945, to include 81 eligible cases previously reported[2] plus 113 subsequent operations. From the 196 consecutive cases five are omitted for the following reasons:

> Three patients from South America had more or less difficulty with English and their "thresholds for speech" are not comparable with the remainder of the group.
>
> One patient left St. Louis before her post-operative hearing tests were completed.
>
> One patient's fenestra closed very soon after the operation and before we had administered our usual speech tests. (Earlier post-operative pure-tone audiometry showed a satisfactory immediate result.)
>
> Three patients gave very erratic and unreliable responses, both to pure-tone and to speech tests. This was noted at the time by our clinical assistants who administered the tests. Two of them are recognized as "psychopathic personalities." They are included in the overall count, but their data have been omitted from some of our graphs and calculations.

Loss for speech before and after operation was measured by Auditory Test No. 9 (recorded spondee words) administered at Central Institute for the Deaf or in the Hofheimer Audiology Laboratory at McMillan Hospital. The same recorded versions of the test were employed throughout, and physical and psycho-acoustic calibrations show the same normal threshold (22 db re 0.0002 microbar) for the two installations. Corrections were made for the slight differences in difficulty of individual lists as determined at the Psycho-Acoustic Laboratory.[5] The general arrangements and specifications of apparatus at Central Institute have been described elsewhere[6] and test facilities of equal or better quality and convenience have been installed in the Hofheimer Audiology Laboratory. The play-back characteristics at both laboratories conform to the specifications of the National Association of Broadcasters.[7]

Pure-tone audiometry by air conduction, and in most cases by bone conduction also, was carried out as part of the clinical routine at McMillan Hospital. These tests were less well standardized than the speech audiometry. The noise level in the audiometry booth was often above desirable limits. Before November 1947 several different audiometers were employed, and many different individuals have conducted the tests. The air-conduction data have been inspected carefully and they confirm the results of speech audiometry although they are less precise. The pre-operative pure-tone audiograms, in addition to the usual tuning fork tests, etc., formed part of the basis of diagnosis of the amount of nerve involvement; however, our graphs and calculations below are based entirely on the results of Auditory Test No. 9 except as specifically noted to the contrary.

This report deals entirely with "immediate" post-operative thresholds for speech. By "immediate" we mean not less than one and not more than four months after the operation. The test sessions were arranged to fall as close to three months after operation as could conveniently be arranged.

RESULTS

The *immediate post-operative losses for speech* are plotted in Fig. 1. The most recent 111 cases (December 1947 to July 1949 inclusive), not previously reported, are indicated by the solid blocks. The previous 80 cases (September 1945 through November 1947), on which a recent report[2] was based, are shown by the open blocks. The trends for the two parts of the series are quite similar, but both the median and the mode moved to slightly lower values in the more recent series.

The recent cases (solid) show a nearly "normal" distribution with median and mode both in the 26-27 db group. The "foot" of the curve includes two cases in the 18-19 db group, and there is a long "tail" of relatively unsuccessful cases extending to the right from 36 db. Nearly the same numerical values were obtained by estimating the loss for speech from the pure-tone audiogram (average for frequencies 500 and 1000 cps). The scatter of the thresholds for pure tone losses was somewhat greater, however, due to the larger standard error of measurement.[2]

For reference an arrow has been placed in Fig. 1 to indicate the generally accepted criterion of a successful result, namely a residual loss for speech of less than 30 db.

The actual distribution shown in Fig. 1. confirms empirically our proposition that *after the most successful fenestration there will remain a hearing loss for speech of about 20 db.*

We have not calculated the standard deviation for the distributions shown in Fig. 1 because the distributions are not normal in shape, but it is obvious that it is of the order of magnitude of ± 4 db if we ignore the "tail" beyond 36 db. This scatter of the nearly normal part of the distribution curve includes three elements enumerated above: *1)* the scatter due to the error of measurement inherent in the test itself, *2)* the scatter of sensitivities of the inner ears of the patients, and also *3)* the variations in actual acoustic conductivity of

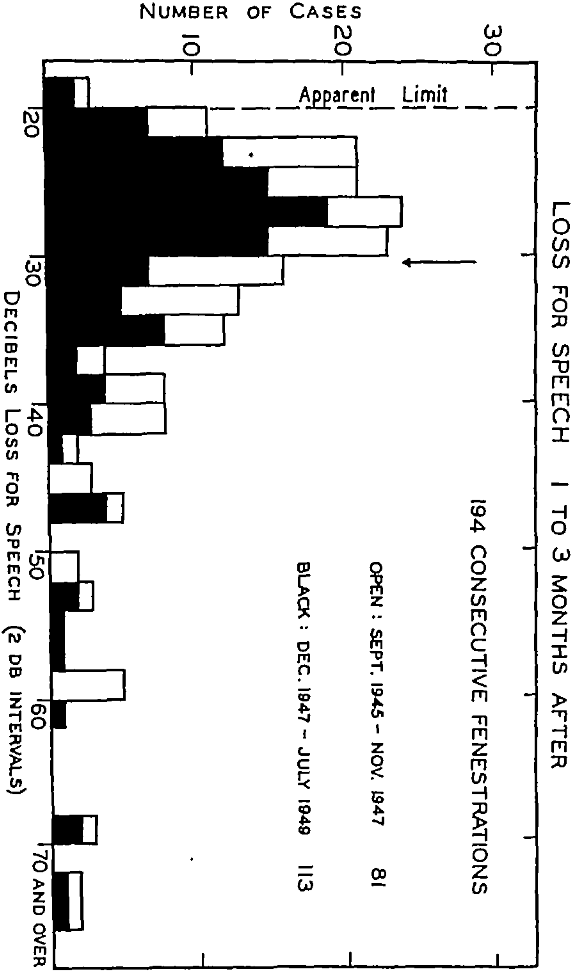

Fig. 1. Test results not corrected for slight differences in difficulty of the No. 9 lists. Two cases with a slight language difficulty, and three who gave erratic responses are included. One successful case operated late in July 1949 is omitted. (This chart was prepared for the meeting of the American Academy of Ophthalmology and Otolaryngology, October 1949,

the fenestras that the surgeon produced. We regard the shape as well as the position of the distribution as excellent confirmation of our first hypothesis.

The second hypothesis, that the maximum possible *gain* from fenestration is about 40 to 45 db, was tested by constructing the scattergram shown in Fig. 2. Each point represents a case. The position of the point on the vertical axis represents the pre-operative loss for speech, the position on the horizontal axis the immediate post-operative loss. Obviously cases that show no gain will lie on the heavy diagonal line, and cases that show 40 db gain will lie on the diagonal line 40 db below the line of no gain. The dashed vertical line indicates a residual loss of 20 db. It is immediately obvious that for cases with initial losses of less than 60 db the gains are all less than 40 db and are limited by the unavoidable 20 db residual loss. For cases with more than 60 db of initial loss the maximum gain is still about 40 db. (We use the round numbers for convenience because the scatter of a few points two or three db beyond these limits may reasonably be ascribed to the unavoidable uncertainties of measurement.) The data, therefore, confirm our hypotheses that the maximum conductive deafness is about 60 db and that the maximum gain from fenestration is 40 (or perhaps 43) db.

It is implicit in our hypotheses that the *degree of fixation of a patient's stapes does not affect his hearing after the operation.* His hearing depends instead on the function of his new fenestra, and, of course, on the amount of nerve deafness that he may have. To examine this point more closely we constructed Fig. 3. Here the cases have been grouped according to the severity of initial loss and the percentage of each group that achieved a given final result has been plotted. The final distributions show nearly the same form for all degrees of initial loss. For all of the groups with initial losses less than 60 db the chances of a good result (*i.e.* less than 30 db residual loss) are the same. For cases with initial losses greater than this the final distributions move systematically to the right. Figure 2 shows that nearly all the severe cases were actually diagnosed clinically as "mixed" deafness.

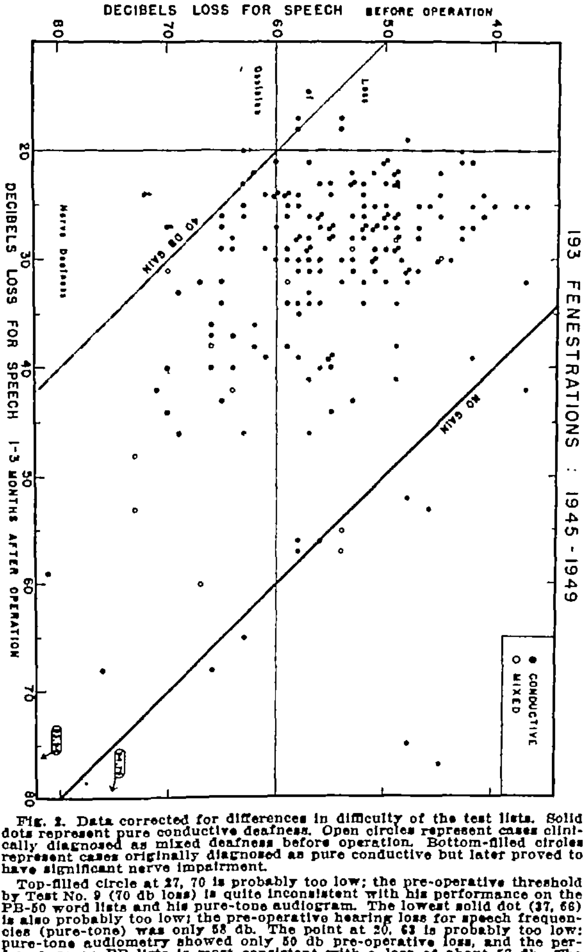

Fig. 2. Data corrected for differences in difficulty of the test lists. Solid dots represent pure conductive deafness. Open circles represent cases clinically diagnosed as mixed deafness before operation. Bottom-filled circles represent cases originally diagnosed as pure conductive but later proved to have significant nerve impairment.

Top-filled circle at 27, 70 is probably too low; the pre-operative threshold by Test No. 9 (70 db loss) is quite inconsistent with his performance on the PB-50 word lists and his pure-tone audiogram. The lowest solid dot (37, 66) is also probably too low; the pre-operative hearing loss for speech frequencies (pure-tone) was only 58 db. The point at 20, 63 is probably too low: pure-tone audiometry showed only 50 db pre-operative loss, and the performance on PB lists is most consistent with a loss of about 57 db. The bottom-filled circle at 24, 72 is certainly too low: another threshold test (for connected discourse), confirmed by the score on PB lists, gave a pre-operative loss of 62 db, not 72. These scattering points that seem to violate our generalizations appear on closer examination to be due to erratic performances on the threshold tests. Also most of the "conductive" cases that have an initial loss of more than 60 db show audiometric indications of a slight nerve involvement.

Two points fall beyond the limits of the chart. One case (78, 94) had very

The final positions of these cases with large initial losses conform to the rule that the greatest possible improvement is a gain of about 40 db.

THE "SURGICAL DEFICIT"

The technical skill of the surgeon should be measured by *1)* the margin by which he fails to provide the best possible acoustic result, *2)* the permanence of the acoustic benefit, and *3)* freedom from undesirable clinical sequelae, such as discharging ears, etc. We are not concerned in the present discussion with the two latter points, in spite of their obvious importance. We do wish to point out that if a surgeon decides to operate on a patient with mixed deafness and achieves the best result that is possible for that patient he should not be blamed if the patient still has his nerve deafness. The total post-operative hearing loss after a technically successful operation may be 35 db instead of only 20 db. The social benefit of the operation for that patient may be very great, as shown by the improvement of his Social Adequacy Index for hearing.[8] If the surgeon fails to recognize in advance the presence of a significant amount of nerve deafness we should criticize not his surgical skill but his ability in diagnosis and prognosis, *i.e.* his *selection of cases.*

We propose the concept of "surgical deficit" to measure the skill of the surgeon in the acoustic plastic surgery called fenestration. His deficit is *the number of decibels by which he fails to achieve the best result reasonably to be expected for that patient.* The surgeon cannot perform the miracle of providing new ossicles. We allow 20 db residual loss on this account, even for the most favorable case. We also allow for any pre-existing hearing loss for speech *due to nerve deafness* that may have been present prior to operation.

Nerve deafness in the presence of conductive deafness may be difficult to measure. Nerve deafness for the lower speech frequencies is not necessarily revealed by a loss of discrimination. Our best present measure is the hearing loss by bone conduction for the speech frequencies. Unfortunately this

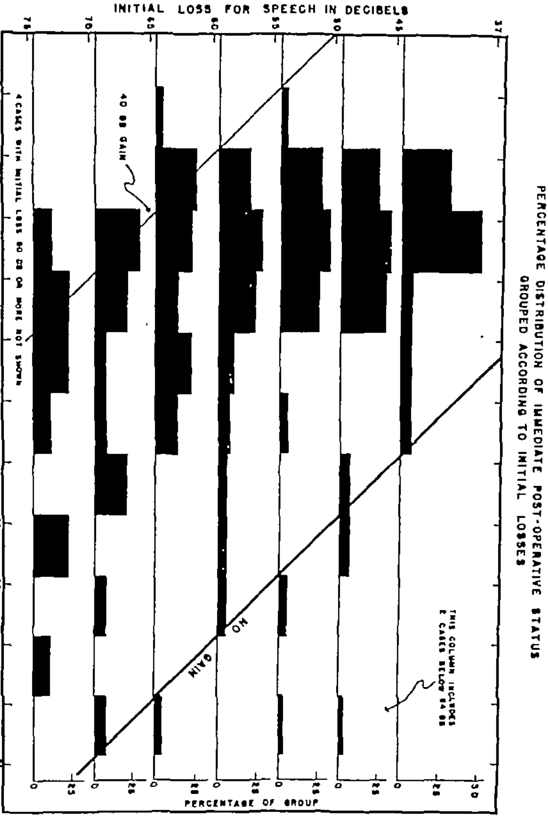

Fig. 3. Same data as in Fig. 2, but without correction to average difficulty. For these percentage distributions the corrections are immaterial. See text for further explanation.

measure is less precise than our measure of air-conduction loss for speech by Auditory Test No. 9. Reasons for the greater uncertainty include:

1) Faulty calibration of many bone-conduction receivers,

2) Possible masking by ambient noise: (Our pure-tone audiograms were taken during routine clinical testing, not in the Audiology Laboratory),

3) Uncertainty as to whether proper masking was used for the contralateral ear. (It may be *impossible* to adjust masking so as to measure the bone conduction threshold of an ear with mixed deafness if the opposite cochlea is normal but is occluded by otosclerosis.)

4) Elevation of the bone-conduction threshold by fixation of the stapes.[4] This elevation may amount to 8 or 10 db for the frequencies near 1000 cps that usually determine the thresholds for speech.

We attempted to allow for these factors by calculating the average pre-operative bone-conduction thresholds (average of 500 and 1000 cps) for the cases operated since October 1, 1947,* that attained the best post-operative results, *i.e.* within 25 db of normal. This group of ears cannot contain any with significant nerve impairment. The average pre-operative bone-conduction hearing loss for this group was 13 db. We considered 13 db apparent loss as "normal" when we estimated the presumable nerve impairment of other cases.

The best result that the surgeon can hope to achieve is to leave the patient with a loss for speech equal to 20 db (loss of ossicles) plus his nerve impairment. Residual loss for speech greater than this is the "surgical deficit." For our most successful cases the deficit is zero. The most common figure is a deficit of 6 or 7 db, both for cases without nerve impairment or with nerve impairment as great as 30 db. *A deficit of 12 db we consider as within reasonable limits.* The most common deficit of 6 or 7 db includes the normal variations in sensitivity of the inner ear, the errors of measurement, and the effects of uncontrollable minor variations in the physical properties of the tissues (such as thickness and stiffness of the skin) as well as factors under the control of the surgeon, such as size and placement of the fenestra and skin flaps, etc., etc. The greater the surgeon's skill (and good fortune) the smaller his deficit will be. At present we consider a deficit of 6 db a "good score." A residual hearing loss of 20 db (a deficit of zero) is a rare affair, like a sub-par "birdie" in golf. A deficit of 12 db is just passable. It lies close to the lower limit of our present "normal" distribution.

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*All of these cases were tested with the same audiometer, although by several different operators.

The complete formula for the surgical deficit is

Deficit = (Residual air-conduction loss for speech) − (20 db) − (residual bone-conduction loss for speech.)

The greatest uncertainty lies in the measurement of the final item.

We do not present in detail the data on surgical deficits. They involve corrections for nerve impairment, and the corrections calculated from the bone-conduction measurements are obviously excessive in several cases (presumably due to errors in the measurements) and lead to impossibly large negative deficits.

EXPLANATION OF SURGICAL FAILURES

The accumulation of cases at the left of the distribution in Figure 1 strongly suggests a "normal" distribution, *i.e.* cases that are fundamentally alike and differ only in random details that are not identified or under control of the surgeon.

In addition to this "normal" random scatter there is the long tail of unsuccessful cases from 35 db onward. Most, although not all, of those in the 32 to 40 db group gave evidence clinically and by bone-conduction audiometry of a mild nerve involvement (see Fig. 2). We have examined with special care, however, the cases with an immediate post-operative loss for speech of more than 40 db. These are failures, real or apparent, that lie entirely outside the range of the normal distribution. There are 30 such cases, 15.7 per cent of the series. Twenty-three of these "failures" had been recognized before operation as cases of "mixed" deafness.

The surgical deficits for 8 of the 23 "failures" with mixed deafness were not over 12 db. The surgeon did as good a job for them acoustically as he did for his average "successful" cases. In six more of these 23 cases the surgical deficit was from 13 to 20 db. These cases are not satisfactory, but neither are they very serious failures; and we should recall the uncertainties of our bone-conduction measurements. For the cases for which we have no bone-conduction measurements we cannot even estimate the surgical deficit, and for several others the measurements are open to more question as they were done before our present audiometer was installed.

Two of the remaining cases with large surgical deficits we may discount in part because in addition to their nerve impairment they gave erratic and unreliable responses during the hearing tests. The patients were characterized by the surgeon as "psychopathic personality." (Erratic responses and personality difficulties may have also contributed to the deficits in another case which is, however, fairly well explained by clearly recognized nerve deafness.)

In five cases there were large surgical deficits of 25 db or more, even when full allowance is made for a recognized and measured nerve deafness. For two of the five cases, which occurred very early (the second and the ninth) in the series as a whole, we have no explanation of the large surgical deficit other than the relative inexperience of the surgeon. In three of the remaining cases some surgical irregularity was noted that gives at least a plausible explanation. The commonest surgical accident (with or without nerve involvement) was a tendency to bleeding at the fenestra. The bleeding may have continued after the wound was closed. In two of the three present cases the fenestra had to be made through otosclerotic bone which bled profusely, and the third was noted at the time of operation for troublesome bone bleeding.

Four more of our cases with no nerve deafness but with large surgical deficits can be explained on purely surgical grounds. In one case the ear drum was torn and failed to heal. It is a matter of common experience that an intact ear drum is essential for a satisfactory result, although the acoustic importance of the intact drum has never been adequately explained. In another case the horizontal canal of the membranous labyrinth was torn and a blood clot was seen in the vestibule. Curiously enough the loss for speech in this ear two months later was only 46 db, an 11 db gain from its pre-operative condition. This is a surgical deficit of only 26 db. *The two other cases were noted at operation for troublesome* bleeding at the fenestra. This is the commonest apparent cause of a large surgical deficit that we have been able to identify: five cases in all.

There remain only three cases of failure with large surgical deficits and one less severe case (surgical deficit of 20 db) for which we have no explanation. One of them, to be sure, was not a case of otosclerosis but of dense adhesive deafness. Two other cases of this type had good results, and we do not know why the fenestration should not have benefited this one also unless dense adhesions remained in the round window niche and somehow interfered with its function. The hearing loss was increased by the operation and the surgical deficit was 32 db. The other three, with surgical deficits of 37 and 27 and 21 db, were typical otosclerosis. Two were pure conductive deafness and the third had only moderate nerve impairment. All three were "perfect operations." Their fenestras are open but their hearing in each case is within 10 db of their pre-operative levels. In these cases it is legitimate to speculate as to the probability of closure of the round window by the otosclerotic process. It is important, however, that *in only three cases (1.6 per cent) in the series is it really legitimate to postulate some such additional purely hypothetical factor.* In all other cases we have at least a plausible and in many cases a quantitative explanation of the immediate post-operative status, whether the result is clinically satisfactory or not and whether the "surgical deficit" is large or small. Only two or three more cases at most could reasonably be added to the "unexplained" list as being insufficiently explained on the basis of nerve impairment.

THE SURGEON'S SCORE

The evaluation of a surgeon as a fenestrator is apparently two-fold: first, what are the chances of a serious surgical failure, *i.e.* a surgical deficit of 20 db or more, due to bleeding, torn labyrinth, torn drum, etc.? For the present the surgeon must also carry the responsibility for the X factor also, whether it is an undiagnosed fixation of the round window or whatever else it may be. Second, what is his mean surgical deficit for cases that are not failures and what is his standard deviation? In other words, what is his expected performance, and what are the chances of any particular case coming within say 5 db of the average? Third (not considered in this paper), what are the chances that improvement will be permanent? Fourth, how good are the surgeon's prognoses? Does he assess accurately the presence and importance of nerve impairment and discrimination loss? These conditions he cannot be expected to alleviate, but he must evaluate them accurately and not mislead himself or his patients as to the probable extent of the benefit of the operation. (It is here that the Social Adequacy Index is helpful.)

We may reckon one kind of rating for a surgeon by noting the incidence of "failures" for which he is clearly to blame or for which no reasonable cause such as nerve involvement, language difficulty, etc., can be found. For example, let us consider a surgical deficit of 20 to 25 db as a simple failure and over 25 db as a serious failure. The 30 cases in the present series with post-operative losses of more than 40 db, discussed above, were grouped in this way. Twelve of them prove to be satisfactory surgical results when allowance is made for nerve impairment, etc., and five more are failures but not serious failures. Thirteen, however, or 6.8 per cent or one in 15, are serious failures.

The incidence of these serious failures in our series is illuminating, however. Six of the 13 occurred in the first 29 cases of the series, between September 28, 1945, and November 1, 1946. One cannot escape the conclusion that, although the surgeon had performed 53 fenestration operations (most of them using the cartilage stopple), and several revisions, before September 28, 1945, his technique was not fully developed until early November 1946. By this time he had performed 165 fenestrations, not counting revisions. Since November 1, 1946, his incidence of serious failures as defined above has been 7 out of 167, or 4.2 per cent.

SELECTION OF CASES

The incidence of cases with severe nerve involvement was much higher in 1945 and 1946 than it has been since. The surgeon has, since 1946 when we developed the concept of the Social Adequacy Index, discouraged from operation more and more strongly all patients with evidence of serious nerve involvement. The nerve involvement is often shown very clearly by a severe loss for high tones by bone conduction and a significant discrimination loss on our monosyllabic word test. Partly as a result of the improved selection cases there are only four cases since December 10, 1948 (the last 52 cases), with post-operative hearing losses as much as 40 db. All four were operated in February, 1949. Three of the four had considerable nerve involvement, and no cases with so much nerve loss have been operated on since.

The importance of selection of cases is shown in Figure 4. Here we have plotted separately the immediate post-operative results for the cases diagnosed clinically before operation as "pure conductive deafness" and as "mixed deafness" respectively. Two cases discovered after operation to have a very significant nerve loss have been transferred from the pure conductive to the mixed category. As before, the solid blocks represent the cases operated since November 30, 1947, and the open blocks those prior to that date.

No attempt is made in this figure to correct for the amount of nerve deafness in the mixed cases. In many of them the nerve loss affected only the high tones and did not elevate the threshold for the lower speech frequencies. That is why some of the most successful cases are in the mixed group. In fact the mixed group is distributed almost evenly from 17 db

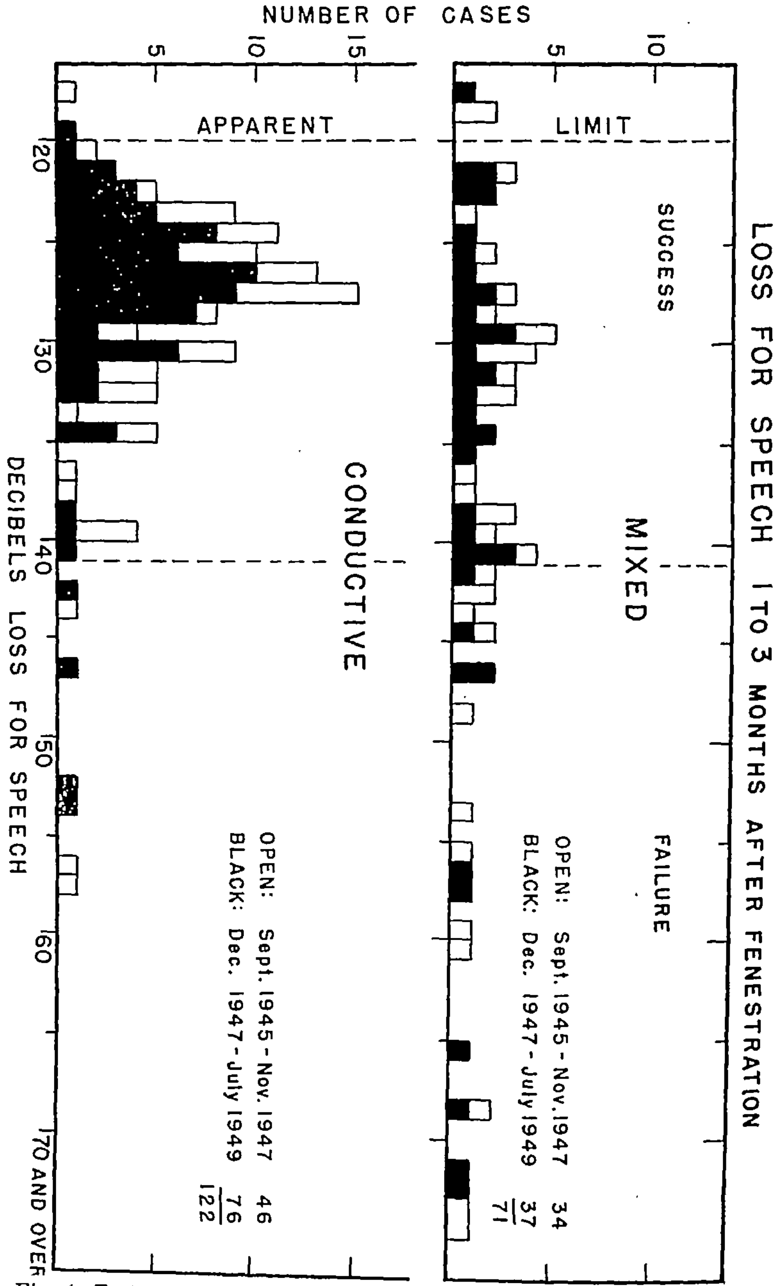

Fig. 4. Test results have here been corrected to average difficulty. Cases are subdivided into conductive and mixed deafness. All three cases with language difficulty are excluded, but the three who gave erratic responses (all with mixed deafness) are retained.

residual loss to complete failure (over 70 db residual loss); but among the 15 cases identified by the surgeon as having the greatest nerve involvement only one had a post-operative loss as small as 35 db.

The 77 more recent cases of pure conductive deafness form distribution centering at 26 db. If we disregard the "tail," *i.e.* cases with more than 35 db loss, the remaining 69 cases form an almost perfect normal distribution with a mean of 26.4 db and a standard deviation just over 3.8 db. These two numbers and his incidence of failures measure the surgeon's skill in providing the best possible immediate acoustic result.

The 46 earlier cases of pure conductive deafness include a "tail" of eight cases with residual losses of more than 35 db. The percentage of such unsuccessful cases is 17 per cent as compared with 9.2 per cent in the recent series. The mean for the remaining 38 cases is 27.1 db with a standard deviation of about 3.8 db. In the more recent series the surgeon averaged a fraction of a decibel better and became a little more consistent in his performance. This is just the sort of improvement that might well be the result of accumulated experience and practice.

The trend of the results with the pure conductive deafness is shown graphically in Fig. 5. Here the results have been averaged in groups of 10 to 15, omitting the failures with post-operative losses of more than 40 db. The size of the groups was chosen arbitrarily to bring the divisions at the beginning of calendar years, vacations, etc. (The "recent series" is the cases operated in 1948 and 1949 plus 2 in December 1947.) There is an abrupt improvement in mean score after the summer of 1947. The fluctuations since that time are not significant, and depend chiefly on whether one, two or three of the less successful cases (30 to 40 db residual loss) happen to fall in a particular period. The general average for 1948 is 26.5 db; for 1949 it is 26.6 db. There is no evidence here that improvement in the surgeon's skill still continues. On the other hand there have been no serious

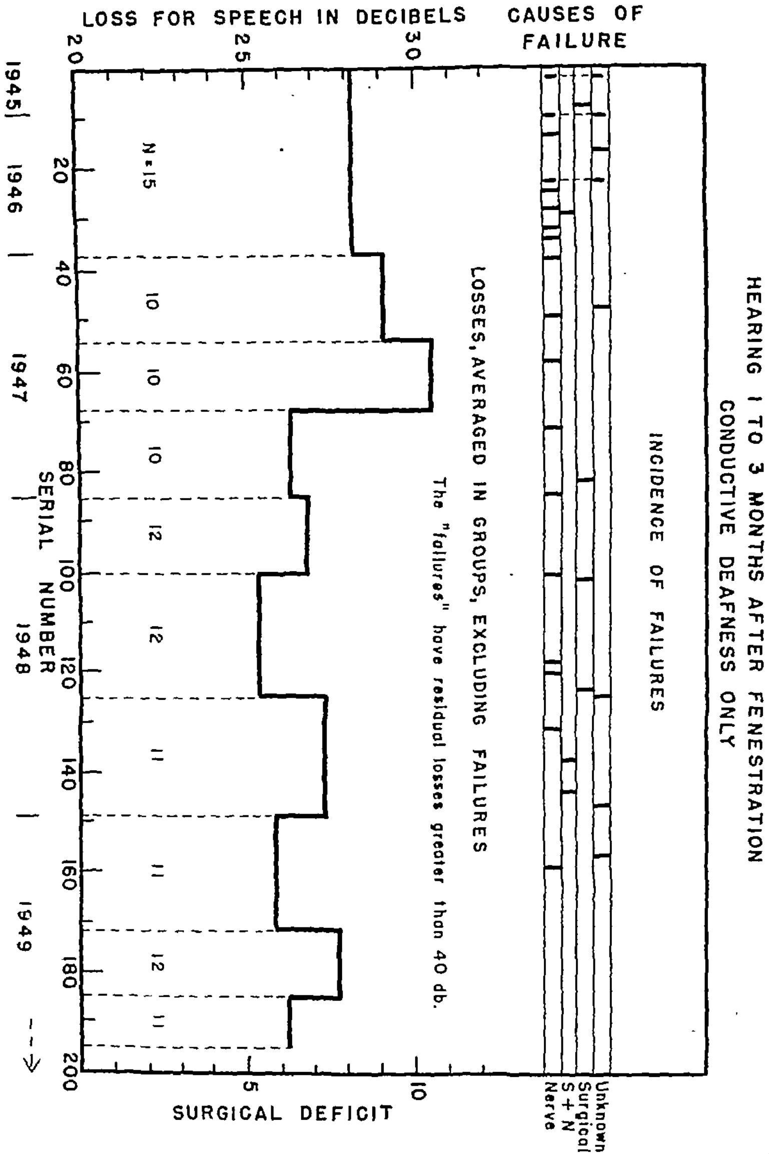

Fig. 5. The breakdown of causes of failure follows the text. "Failure" means an immediate post-operative hearing loss for speech of more than 40 db, and includes cases of nerve deafness, surgical accident and unknown causes. One case (with a "psycopathic personality") whose responses were unreliable has been omitted. Two other erratic cases, in which nerve deafness was obvious and severe, have been included. As far as possible each case has been classed in the single category that seems to be the chief cause of the failure, but six are divided between two categories. See text for explanation of average losses of the successful cases of conductive deafness.

failures since February 1949. Experience and good selection of cases probably account largely for this; but even with the greatest skill and care we must expect sooner or later to encounter more failures from the "unknown" causes. It is evident, however, that the immediate acoustic result for future members of this series can be predicted with considerable assurance and fair accuracy. Indeed, recently the surgeon has been in the habit of indicating on his patient's chart the expected S.A.I. He has been consistently accurate to within ± 5 S.A.I. points. The expectations for the permanence of their gains we shall consider at a later time when more long-term data are available.

SUMMARY.

The hearing losses for speech have been measured before and shortly (about three months) after 195 consecutive fenestration operations.

The data are compared with the results of an analysis of known physical and psycho-acoustic factors involved. They are in excellent agreement, and support the following propositions:

1) The best result to be expected following fenestration is a residual hearing loss for speech of 20 db. This minimum loss depends primarily on absence of the ossicles.

2) The residual hearing losses of successful cases scatter at random about a mean. The scatter is due to

  a) The "error of measurement" of the hearing test:

  b) Normal variations in the acuity of the inner ear;

  c) Variations in the physical properties of the patients' skin flaps and other tissues, and uncontrolled minor variations in surgical procedure;

3) The maximum possible *conductive* hearing loss for speech is about 60 db. When speech is more than 60 db above normal threshold it can be heard by bone conduction. Losses greater than 60 db therefore mean that more or less nerve deafness must be present. This is confirmed by bone-conduction audiometry.

4) Nerve deafness is not alleviated by the fenestration operation. The *maximum gain* that can be expected from fenestration is therefore 60 db – 20 db, *i.e.* 40 db.

5) The residual hearing to be expected after fenestration depends on the amount and character of *nerve* impairment (if any) but not on the amount of pre-operative *conductive* deafness.

The number of decibels by which a patient's immediate post-operative hearing loss for speech exceeds the unavoidable minimum we call the "surgical deficit." For pure conduction deafness the deficit is the excess above 20 db residual loss. For mixed deafness additional allowance is made for the "nerve" component of the deafness. The latter is at present best measured as the hearing loss by bone conduction. Several difficulties attending such measurements are discussed.

The mean post-operative hearing loss for our 69 cases of pure conductive deafness operated since December 1, 1947, excluding definitely unsatisfactory cases with final losses greater than 35 db, is 26.4 ± 3.3 db.

The incidence of failures, the mean surgical deficit for the remaining cases, and its standard deviation are three appropriate measures (among others) for evaluating a surgeon's technical proficiency. If accurate estimates of nerve impairment are available we can, from these measures of the surgeon's skill, make prognoses of known probability. Thirty (15.7 per cent) of our series had an immediate post-operative loss for speech of more than 40 db. Fourteen of these "failures" are due largely or wholly to the presence of significant nerve impairment.

Twelve failures (6.3 per cent of the series) are due to known or probable surgical "accidents," usually bleeding into the fenestra as from otosclerotic bone. Only three failures (1.6 per cent of the series) are completely without explanation.

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A STUDY OF DELAYED BLEEDING FOLLOWING REMOVAL OF THE TONSILS AND ADENOIDS.*†

ARTHUR J. COOMBS, M.D.,
Chicago, Ill.

Hemorrhages following the removal of the tonsils and adenoids are often embarrassing to the otolaryngologist and may even be fraught with grave consequences. Anything that may be done to lessen the occurrence or alleviate the severity of delayed bleeding will ease the physician's task, lighten his responsibility and enhance his reputation in the sight of his patients. A study of the causes of postoperative bleeding was undertaken in the hope that something worthwhile might be discovered.

Statistics showing the incidence of hemorrhages varied considerably among equally competent surgeons. A review of the author's own records indicated that apparently a record was made on a patient's chart only in the more or less severe cases. No report of slight bleeding was found, although it was easy to recall that such cases had occurred. Fox and West[1] have stressed the importance of persistent follow-up reports on all cases, even using the telephone and mail service when necessary, otherwise many cases of bleeding will be missed. Due to such previous laxity, it was impossible to establish an accurate control series for comparison with the experimental series that are to follow. Possibly some authors distinguish between hemorrhages and slight to moderate bleeding which causes little inconvenience and stops spontaneously within a few minutes. In this case such statistics would indicate more favorable results.

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*From the Department of Otolaryngology, St. Luke's Hospital, Chicago, Ill.

†Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 2, 1950.

HISTORICAL.

Since 1940, a number of articles have appeared in the literature on the relationship of hypoprothrombinemia and the salicylates, more particularly acetylsalicylic acid. Attempts to correct this deficiency by the administration of vitamin K have been reported both favorably and unfavorably by numerous investigators. In this year, Elliott, Isaacs and Ivy[2] produced a dietary prothrombin deficiency in the rat and showed that a complete return to normal could be obtained by the administration of vitamin K. In 1941, Kark and Souter[3] reported that spontaneous bleeding might occur from pathological surfaces, such as wounds, ulcers, etc., when the prothrombin level in the blood is down to 35 per cent and a hemorrhagic diathesis may occur if it is lowered to 15 to 20 per cent. They further state that the intravenous administration of vitamin K "controls hemorrhage from hypoprothrombinemia in one and one-half to three hours while the blood prothrombin level rises rapidly and reaches a normal level within 24 to 48 hours." Other authors, especially Link[4] and his co-workers, have demonstrated a parallelism between the hypoprothrombinemia produced by hemorrhagic sweet clover disease and salicylic acid. Shapiro, Redish and Campbell[5] *state that "the action of salicylate is apparently identical with* that of the anticoagulant, dicumarol, but less effective," and that acetylsalicylic acid is more potent in this respect than sodium salicylate.

Clinical reports have been appearing since then showing a close correlation between the use of salicylates and bleeding that occurs after the removal of tonsils and adenoids. Among the first of these was an article by Singer[6] that pointed out the greater frequency of postoperative hemorrhages in this country compared to European countries was due to the prevailing custom of using aspirin here compared to aminopyrine over there. In the salicylate medication of rheumatic fever patients, Rapoport, Wing and Guest[7] state that there is a "marked prolongation of the prothrombin time of the blood." Meyer and Howard[8] confirmed this and showed that the concomitant administration of vitamin K prevented the development of a hypoprothrombinemia. Fox and West,[1] as well as others (Stone[9] and Howell[10]), have shown that vitamin K is obtained physiologically from the normal intestinal flora and stored in the liver. The absence of hepatic disease is, therefore, a primary consideration in all of the reports on salicylate induced hypoprothrombinemia. Other reports advising the simultaneous use of vitamin K with acetylsalicylic acid have shown marked improvement in the reduction of postoperative hemorrhages. Fox and West[1] have attacked the accuracy of some of the reports on the basis of "too little laboratory data." They have concluded that there is no protective value in using vitamin K postoperatively as hypoprothrombinemia is not the cause of delayed tonsillar hemorrhage. They feel that the high incidence of bleeding following the use of chewing gum containing aceylsalicylic acid is due probably to some local effect on the wound. Ersner[11] and associates, in 1934, indicated that saliva mixed with blood becomes a potent factor in decreasing the coagulation time and that the presence of aspirin in the saliva delayed the formation of a clot.

The rôle played by vitamin C as well as vitamin K in preventing hypoprothrombinemia has been stressed by Neivert[12,13] and others.

PHYSIOLOGY.

The classical theory of blood coagulation established in the latter part of the last century is still generally accepted among most investigators. Prothrombin, thromboplastin and calcium unite to form thrombin. Thrombin and fibrinogen then react to produce fibrin or the clot. Anything that will lower the prothrombin content of the blood plasma thus will interfere with the normal process of blood coagulation. It is with this phase that the otolaryngologist is most concerned as a means of preventing delayed postoperative bleeding.

CLINICAL PROCEDURES.

In order to make an accurate study of postoperative bleeding, every case was operated upon by the author and followed at least 10 days by routine postoperative office call or by a report over the telephone. All children were done under general anesthesia and in most cases both the tonsils and adenoids were removed, although in some only one or the other was done. The tables do not differentiate in this matter. The adults were done under local anesthesia, employing 1 per cent procaine hydrochloride with eight drops of epinephrine hydrochloride to the ounce for better hemostasis. In removing the adenoids, a LaForce adenotome was used first in the midline and then again on each side, being careful to keep the blade from injuring the cushion of the Eustachian tubes. The area was inspected and any residual lymphoid tissue removed with a punch forceps. The tonsils were removed by blunt dissection and snare, sharp dissection being employed only in the case of marked fibrosis of the capsule. In general, all primary bleeding was controlled by sponge pressure, but catgut sutures or ligatures were used whenever necessary.

For the purposes of this paper only delayed bleeding is considered, that is, bleeding which occurred only on the second postoperative day or later. Also, all cases of bleeding, no matter how slight, are recorded, and on this account the term "hemorrhage" will not be used except in case of a direct quotation where the patient used the term in his report.

In the first series, comprising 164 cases, 10 mg. Synkayvite were given with meals three times daily and one tablet of Aspergum was ordered about 10 minutes before eating. In this series there were 12 cases (7.31 per cent) of bleeding, only five requiring medical attention (3.05 per cent) (see Chart 1).

In the second series, comprising 310 cases, 10 mg. Synkayvite, 250 mg. ascorbic acid and one tablet of Aspergum were given three times daily with meals. In this series there were 32 cases of bleeding (10.32 per cent) but only three that required medical attention (0.97 per cent) (see Chart 2).

In the third series comprising 179 cases there were 25 cases of bleeding (13.96 per cent), of which 13 (7.26 per cent) required medical attention (see Chart 3).

CHART 1.
SYNKAYVITE AND ASPERGUM.

No.	Name	Age	Date Oper.	Postoper. Day of Bleeding	Remarks
1	E. P.	5	9/21/45	5th	Bled from nose freely.
2	J. R.	8	10/12/45	5th	Profuse adenoid bleeding; postnasal tampon used.
3	F. K.	6	2/16/46	6th	Moderate bleeding
4	K. K.	32	2/18/46	6th	Moderate bleeding off and on; small clot removed on the 8th day.
5	E. B.	38	4/ 1/46	5th	Slight bleeding.
6	L. M.	5	5/ 9/46	8th	"Had a gush of blood from the nose lasting one minute. Many nosebleeds all of life."
7	S. S.	5	5/ 9/46	7th	Adenoid bleeding rather severe.
8	B. W.	11	5/14/46	6th	Slight spotting on pillow; did not awaken.
9	J. O.	6	7/ 1/46	7th	Very slight bleeding; "like a tooth pulled."
10	A. G.	5	7/ 5/46	8th	Adenoid bleeding; sponge pressure used.
11	G. S.	27	7/ 9/46	6th	Bleeding tonsil fossa; sponge pressure used.
12	D. M	8	7/11/46	7th	Severe adenoid bleeding; postnasal tampon used.

Total number of cases in this series was 164. Ten mg. Synkayvite were given three times a day and Aspergum before meals. Cases requiring medical care were five. Cases stopping spontaneously were seven.

CHART 2.

SYNKAYVITE, ASCORBIC ACID AND ASPERGUM.

No.	Name	Age	Date Oper.	Postoper. Day of Bleeding	Remarks
1	E. K.	29	11/15/46	5th	Reported as "slight hemorrhage."
2	J. C.	4	11/18/46	7th	Severe adenoid bleeding; sponge pressure used; took only two doses of medication.
3	D. G.	4	11/22/46	3rd	Moderate adenoid bleeding.
4	N. K.	14	11/26/46	5th	Slight spotting.
5	B. B.	7	11/26/46	5th	Slight adenoid bleeding.
6	J. R.	4	1/14/47	4th	"Slight bleeding. Lasted only a few minutes."
7	T. F.	8	4/21/47	9th	Adenoid bleeding; postnasal tampon used.
8	N. K.	4	5/ 5/47	3rd	Adenoid bleeding.
9	P. O.	10	6/10/47	5th	Moderate bleeding.
10	A. K.	8	6/ 6/47	3rd	Very slight bleeding.
11	D. C.	6	6/17/47	4th	Slight adenoid bleeding.
12	D. S.	4	6/27/47	8th	Slight adenoid bleeding.
13	J. T.	4	7/11/47	5th	Slight bleeding.
14	R. K.	18	8/25/47	6th	Bleeding tonsil fossa; sponge pressure used.
15	I. P.	5	10/ 6/47	6th	Moderate bleeding from nose.
16	R. F.	6	10/24/47	8th	Slight adenoid bleeding.
17	M. M.	4	10/31/47	11th	Slight bleeding.
18	M. J.	4	11/ 4/47	8th	"Expectorated small clot from the throat."
19	R. B.	3	11/17/47	5th	"Expectorated a large clot from the throat."
20	R. B.	6	11/24/47	6th	Bleeding from nose.
21	W. H.	4	11/28/47	5th	Slight bleeding.
22	B. S.	11	12/ 5/47	5th	Slight adenoid bleeding.
23	D. B.	7	12/15/47	6th	Moderately severe bleeding.
24	B. B.	7	12/29/47	8th	Adenoid bleeding.
25	K. C.	5	1/16/48	9th	Slight adenoid bleeding.
26	J. S.	5	1/17/48	4th	Moderate adenoid bleeding.
27	J. G.	5	3/ 5/48	7th	Moderately severe adenoid bleeding.
28	J. C.	7	3/26/48	4th	Moderately severe adenoid bleeding.
29	S. M.	4	4/30/48	6th	"About a teaspoonful from the nose."
30	T. H.	5	6/24/48	5th	"Slight bleeding from the nose for about two minutes."
31	J. S.	5	7/ 1/48	6th	Slight bleeding off and on seventh and eighth days.
32	M. D.	6	7/16/48	7th	Slight bleeding from the nose.

There was a total of 310 cases in this series. All had 10 mg. Synkayvite three times a day, 250 mg. ascorbic acid three times a day and Aspergum before meals.

Cases requiring medical attention were three, the other 29 stopped spontaneously.

CHART 3.
ASCORBIC ACID AND ASPERGUM.

No.	Name	Age	Date Oper.	Postoper. Day of Bleeding	Remarks
1	B. J.	5	9/17/48	7th	Severe adenoid bleeding; postnasal tampon and three transfusions.
2	C. P.	6	10/29/48	2nd	Severe adenoid bleeding; postnasal tampon.
3	S. K.	7	11/ 1/48	3rd-8th	Moderate adenoid bleeding.
4	A. Q.	4	11/ 3/48	9th	Severe adenoid bleeding; postnasal tampon.
5	J. G.	4	11/26/48	5th	"Very slight bleeding for a few seconds."
6	B. D.	6	1/10/49	8th	"Bled about a teaspoonful."
7	P. B.	6	1/21/49	6th	Small amount of bloody emesis.
8	K. P.	5	2/11/49	6th	Slight bleeding.
9	C. K.	6	3/ 1/49	8th	Severe adenoid bleeding; postnasal tampon.
10	C. S.	12	3/28/49	11th	Severe adenoid bleeding; postnasal tampon.
11	T. S.	10	4/12/49	6th	"Slight nosebleed."
12	H. S.	4	4/20/49	9th	Slight spotting on the pillow.
13	E. R.	3	5/ 3/49	3rd	"Slight nosebleed."
14	J. L.	6	5/ 5/49	3rd	Bleeding tonsil fossa; sponge pressure.
15	M. K.	4	5/16/49	7th	Severe adenoid bleeding; sponge pressure.
16	K. R.	9	5/20/49	4th-6th	Moderate bleeding tonsil; two days later adenoids.
17	L. H.	5	5/27/49	7th	Severe adenoid bleeding; postnasal tampon.
18	W. S.	5	6/17/49	4th	Adenoid bleeding; sponge pressure.
19	A. D.	7	6/17/49	6th	Slight adenoid bleeding.
20	J. W.	3	6/24/49	6th	Severe adenoid bleeding; postnasal tampon.
21	M. S.	4	7/ 6/49	6th	Slight spotting from nose.
22	K. Y.	8	7/ 8/49	5th	Moderately severe adenoid bleeding; sponge pressure.
23	P. M.	8	7/ 8/49	8th	Moderately bleeding tonsil fossa; sponge pressure.
24	W. H.	6	7/18/49	3rd	Adenoid bleeding about 10 minutes.
25	L. M.	11	7/22/49	6th	Moderate adenoid bleeding; sponge pressure.

There were a total of 179 cases in this series. Thirteen required medical attention; 12 cases stopped spontaneously. All were given 250 mg. ascorbic acid three times daily and Aspergum before meals.

COMMENT.

The one factor common to each of the series was the use of Aspergum (chewing gum with approximately 3.5 gr. acetylsalicylic acid). Livingston and Neary,[14] in a small series of cases, were unable to find any evidence that this would produce a hypoprothrombinemia. Fox and West[1] are inclined to agree with this and think that late tonsillar bleeding is due to some local effect of the aspirin on the wound. A contrary view is expressed by Neivert[13] when he says "most assuredly such a rise (elevation of prothrombin time) will occur eventually," as shown by his experimental studies on normal individuals.

The synthetic vitamin K used is commonly known as Synkayvite and has the formula 2-methyl-1,4-naphthaquinone. It was given in the first and second series to see if it might materially reduce the incidence of postoperative bleeding. As mentioned previously, no attempt had been made to keep as accurate a record of bleeding previously to this study when acetylsalicylic acid was the only form of medication employed, so no true control can be established. The fact that bleeding did occur seriously enough in 3.05 per cent of the cases to require medical attention in order to control it indicates that most certainly vitamin K by itself will not prevent delayed secondary bleeding.

In the second series of cases ascorbic acid was given in conjunction with the vitamin K. It is well known that ascorbic acid is beneficial in most body healing processes. Where a patient is so severely handicapped in the normal ingestion of food, as in the case of a tonsillectomy, the nutrient value of ascorbic acid on the granulating surface might be important in reducing the incidence of bleeding. Although the overall occurrence was higher than with vitamin K alone, there were many cases of slight bleeding, so that the number requiring medical attention was just under 1 per cent. Thus, it would seem reasonable to advise the use of ascorbic acid in conjunction with vitamin K as a probable beneficial measure in cases of tonsillectomy.

In the third series, ascorbic acid and Aspergum alone were used. The overall bleeding was highest in this group, 13.96 per cent, and the serious ones requiring medical attention amounted to 7.26 per cent. By comparing this group with the first, then, it is possible to make a reasonable conclusion that the use of vitamin K reduces the overall percentage of bleeding from 13.96 to 7.31 and the serious ones from 7.26 to 3.05. Since the use of Aspergum was common to all three series, any local action on the wound surfaces that would predispose to bleeding would be nullified for statistical purposes and would not be an important factor. Of greater importance in this clinical study is the effect of vitamin K and more particularly the simultaneous administration of vitamin K and ascorbic acid.

A study of Chart 4 shows a compilation of the days during which postoperative bleeding occurred. It is readily seen that the critical period lies between the third and eighth days, although it may take place at any time through the eleventh day, the greatest frequency being on the fifth and sixth days. Three cases bled on more than one day. It is during this

CHART 4.

Day of Post-operative Bleeding	Number of Cases
2nd	1
3rd	6
4th	5
5th	14
6th	17
7th	10
8th	11
9th	4
10th	0
11th	2

In the above computation one case bled on the third and eighth days, another on the fourth and sixth days, and a third on the sixth, seventh and eighth days and are listed accordingly in the second column.

period that the granulation tissue is forming between the raw surface and the exudative membrane that forms over it. As this membrane its gradually displaced and sloughs away a capillary bed or an individual vessel may become exposed and eroded, thereby starting the bleeding.

Predisposing factors that may increase the incidence of bleeding are the usual blood dyscrasias, congestion of granulations due to the presence of infections or incompletely resolved recent acute exacerbations of respiratory illnesses, dietary deficiencies, particularly the proteins and vitamins, hypertension and local trauma to the tissues, such as might be caused by certain types of food. Any case of suspected blood dyscrasia should be carefully checked by appropriate blood studies, not depending upon the results of the usual coagulation or bleeding time which are taken routinely. The determination of the prothrombin time will afford more accurate information and should be done whenever possible in the preoperative examination of the patient. The operation should never be done in the presence of an acute infection or even in a recent one unless adequate time has been allowed for complete resolution to take place. In questionable cases chemotherapy or the administration of certain of the antibiotics may be given preoperatively and for a day or two postoperatively. McGovern[15] recommends the use of sulfathiazole chewing gum, while Fox and West[1] advocate the use of lozenges containing benzocain and tyrothricin — tyrozets — for their local action in keeping down infection and the latter also for relieving discomfort. These might well be used in tonsillectomies for this purpose, but it is difficult to see how they would be of any benefit as far as the adenoids are concerned. In the author's series covering 653 cases the incidence of bleeding from the adenoids was far greater than from the tonsil fossae.

CONCLUSIONS.

The preponderance of evidence in the literature indicates that salicylates have a depressant action on the formation of prothrombin and thus predispose to postoperative bleeding.

Synkayvite given postoperatively exerts a beneficial effect in lowering the incidence of bleeding.

Synkayvite and ascorbic acid given simultaneously exert a still greater effect in reducing this incidence.

The critical period for bleeding to occur ranges from the third to the ninth days, with most occurring on the fifth and sixth days.

Careful preoperative examination including appropriate blood studies and choosing a time free from recent infections are other important factors in selecting cases for operation.

SUMMARY.

A report of 653 consecutive cases of tonsillectomy and adenoidectomy was made to study the incidence of postoperative bleeding.

Three series of investigations were made to determine if acetylsalicylic acid, Synkayvite and ascorbic acid might have a bearing on the subject.

A study of the time for the occurrence of postoperative bleeding was made.

30 North Michigan Avenue.

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BOOK REVIEW

Bronchography. By Eelco Huizinga, M.D., and G. J. Smelt, M.D., Department of Oto-Rhino-Laryngology, University of Groningen. 270 pages with 146 illustrations. Assen, Netherlands: Van Gorcum & Co., Ltd., 1950. Price: Hfl 39 (Dutch currency; about $10.00 in American funds).

This is an excellent and detailed story of bronchography. The authors devote the first part of this book to the anatomy and physiology of the bronchial tree. They treat the subject comprehensively.

General considerations of bronchography with indications and contraindications compose Chapter 3, and a valuable discussion of the Roentgen anatomy of the normal bronchogram follows.

The discussion of the interpretation of bronchograms with cases and many excellent illustrations is very valuable.

The last chapter is a detailed description of the authors' technique in bronchoscopy and the bibliography is complete.

A very valuable book for any who are interested in diseases of the chest and in endoscopy. T. E. W.

FURACIN SOLUBLE DRESSING NOW AVAILABLE IN NEW TWO-OUNCE TUBE.

New packaging of Furacin Soluble Dressing in a two-ounce collapsible tube is announced to the profession by Eaton Laboratories, Inc., of Norwich, New York. This tube is in addition to the present packaging of the drug in four-ounce, one-pound and five-pound jars.

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Furacin Soluble Dressing is a yellow, water-soluble ointment. It is prescribed for the prophylaxis or treatment of infections of wounds, severe burns, cutaneous ulcers, pyodermas and skin grafts. In addition to its availability as Furacin Soluble Dressing, Furacin is supplied, on prescription, as Furacin Solution (N. N. R.), Furacin Anhydrous Ear Solution and Furacin Vaginal Suppositories.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Illinois Hotel, Bloomington, Ill., on April 21, 22 and 23. Dr. A. E. Braley, New York City, N. Y., will give several lectures on "Diseases of the Eye." Dr. Louis Clerf, Philadelphia, Pa., will discuss "Laryngeal Diseases." Dr. Louis Ostrom, of Rock Island, Ill., and Dr. H. Middleton, of Alton, Ill., will also give papers. For further information address the Secretary, Dr. Philip R. McGrath, 843 Jefferson Building, Peoria, Ill.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

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Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

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Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been *omitted from this list for brevity.*

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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THE
LARYNGOSCOPE.

VOL. LX APRIL, 1950. No. 4

OTITIS MEDIA WITH EFFUSION — A CHALLENGE TO OTOLARYNGOLOGY.*

GORDON D. HOOPLE, M.D.,
Syracuse, N. Y.

Otitis media with effusion is a common condition. Often present both in children and adults, it presents a challenge to otolaryngology. This challenge includes the etiologic, diagnostic and therapeutic aspects of this condition. It might even include the terminologic angle. It has been designated by many terms, none of which is satisfactory. An accurately descriptive term would be welcome. Otitis media with effusion *is used in this presentation simply because it calls attention* to the presence of fluid in the middle ear and not because it is an ideal term. To be certain, however, about the subject under discussion, it should be stated it is that condition in which the middle ear is filled with sterile fluid which may vary from a thin serous character (the most common) to a heavy, thick mucus mass. It is not related to the suppurative otitis medias except as it can be artificially created by the use of the biochemicals. The fluid may remain in the ear for only one day, or it may persist for many years.

There is abundant evidence that the diagnosis of this condition is often missed. Some personal experiences with over-

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*Read at the meeting of the American Academy of Ophthalmology and Otolaryngology, March 20, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 20, 1950.

looked patients in both Army and civilian life were cited when this subject was presented at the 1949 meeting of the American Academy of Ophthalmology and Otolaryngology and bear no repetition here. For years, teachers and textbooks alike have stressed, in the diagnosis of this condition a fluid level line. This is a snare and a delusion, for if one depends upon the observance of a fluid level line as a diagnostic aid in this condition more than nine out of every 10 cases will be missed. This is because the full tympanum and, much more often than not, the full mastoid as well, is 10 times as prevalent as the the ear which has only a level line. In none of our current textbooks, literature or teaching does one find these relative figures. It is not realized by most otologists and consequently is not taught to the younger ones.

Over the years in a general (not solely otologic) otolaryngologic practice, patients suffering with otitis media with effusion, percentage-wise, will total between 3 per cent and 4 per cent of all patients who visit the otolaryngologist. In view of this frequency and the fact that its diagnosis is often missed, the etiologic and diagnostic factors of this condition should frequently be reviewed.

ETIOLOGY.

Much is known of the etiology of otitis media with effusion but not all. It is the unknown which presents the challenge. When the unknown has become known we may have some therapeutic aids which are lacking now.

What do we know of the etiology of fluid in the middle ear? In general, it may be said that anything which will contribute to the Eustachian tube swelling and closure can be a contributing cause. More often than not an acute upper respiratory infection, abetted or not by the presence of local structural abnormalities or complicating factors, will precede the condition. These abettors are well known and require no detailed listing. (Dental malocclusion should perhaps be mentioned as it is sometimes overlooked.) If they are present, fluid is more apt to appear in the middle ear than if they are not; if one-sided, they may be the cause of the presence of fluid in the homolateral ear; however, a blocked Eustachian tube is not, in itself, the whole answer to the problem, for if it were, every closed Eustachian tube would be accompanied by fluid in the middle ear. Why, then, when the Eustachian tube closes does fluid form in some ears and not in others?

Robison[1] has advanced the explanation that the presence or continuance of fluid in the ear is due to a blockage in the lymphatic glands which drain the middle ear region. Lately, our attention has been called to the sizable percentage among all who are afflicted with fluid in the ear, of those who are allergic. Jordan[2] puts it in the neighborhood of 70 per cent. Williams,[3] stating he believes the allergy in some to be physical in nature, intimates this additional type of allergy is present more often than is suspected. Both the regional blockage theory and the goodly number of allergic patients suggest that conditions outside the ear may be factors for the presence or continuation of fluid in the ears. It is in this area that we, as otolaryngologists, may have an Achilles heel. Focusing our attention on the ear and its neighboring parts, we may fail to recognize the presence of general conditions or disorders which may play a significant part in the condition under discussion. Anything which abnormally affects the water balance of tissues in the body can adversely affect the course of otitis media with effusion. Allergy, a known offender in this regard, has already been mentioned. Endocrine disorders of several types, not excluding pregnancy, are potential sources of trouble. Cardiac insufficiency and certain forms of cardio-vascular-renal disease have been known to be causative agents in the persistence of fluid. Hilger[4] has recently called our attention to autonomic nervous system disorders, many times psychic in origin, as disturbers of tissue fluid balance. All of these may underlie some of our cases of otitis media with effusion. Their possible presence challenges our diagnostic acumen. Unless we ferret them out, when they are present, we are doomed to a greater percentage of failure to cure than should be our lot.

Senturia[5] stresses the presence of a negative pressure in the tympanum as the mechanism by which fluid is drawn into the ear. This undoubtedly is present in the instances where there is fluid but not all ears with a negative pressure develop fluid and, apparently, many have open Eustachian tubes long before fluid disappears from the ear. During World War II, when there were so many ruptured membrana tympani, an unusual opportunity was afforded for inspection of the middle ear mucosas. The majority were thin and dry. A small number, apparently without infection, were thick and moist. The cause behind the difference? One can speculate and run through a gamut of ideas from a poor development of the tympanal mucosa according to Witmaack's theory (advanced years ago) to the current emphasis on allergic mucous membranes. The cause of the difference, we can temporarily dismiss, but the fact that there is a difference, we must accept. This suggests that some ears under a given set of circumstances may develop fluid while others will not. Etiologically, the cause of the difference in mucosas does not matter, for when the patient presents himself fluid is already there. Therapeutically, however, knowledge of the cause of the difference may mean much, for that knowledge may hold the secret which will bring about an effective cure.

The two preceding paragraphs have stressed the possibility of a general condition as an etiological factor in the prevalence and persistence of otitis media with effusion. This must not be construed as an emphasis which excludes the local abettors mentioned above. Were it not for the latter, many of these "moist" ears would never develop fluid. That local pathology can be etiologic and may be a cause for persistence must never be forgotten. In protracted cases, for instance, one must repeatedly look at the nasopharynx lest a developing malignancy be overlooked.

Eagle[6] and Tobey[7] in recent years have published reports which mention a recent marked increase in the incidence of otitis media with effusion. Politzer[8] described the condition accurately and wrote at length of it in his textbook, which suggests it was well known and often recognized in the latter part of the last century. The present author has noted only a 20 per cent increase in the incidence of fluid in the ear since the advent of the biochemical treatment of purulent otitis media. (With the use of this therapy and consequent disappearance of pus cells, the ear can look and act like one which has never gone on to suppuration.) The greater part of the 20 per cent increase is to be found in children. It goes without saying that there must be thousands upon thousands of such ears which never come to the attention of the otolaryngologist, for few pediatricians or general practitioners test the hearing following the use of the biochemicals for suppurative otitis media, and fewer would recognize the presence of the condition if they happened to look into the ear.

Fluid in the ears may develop after trauma or in aerotitis. These cases are few in number and do not present the problem of persistence in like degree to those caused by other etiological factors.

The effusion has been known to continue in its fluid state and remain present in an ear for years. Sometimes after one, or repeated attacks (and these occur), there are resultant adhesions which bind the drum and ossicles to each other and the promontory, with a consequent hearing loss which varies with the location and strength of the adhesions. Why some ears will clear in a reasonable period, others have a persistence of fluid for months or years, and still others terminate with adhesive processes is not exactly known. As we learn more about tissue chemistry, general metabolic processes, electrolytic balances, etc., we may arrive at the final answers.

PATHOLOGY.

There are two types of fluid which may fill the ear. One is of a thin serous character and a negative pressure in the middle ear is the cause of its presence. It is a transudate. Lowy,[9] Robison and others have pointed this out. The second is mucus in character and is exudative. When present, it is usually mixed with the serous transudate. The degree to which mucus is present is indicative of the mucosal response to inflammation. The amount, as intimated, is variable. There may be none and it is conceivable the entire fluid response of an ear may be mucus in character. While there may be many variations of the mucus contents of the fluid, clinically an ear is either a "serous" ear or a "mucus" ear, the former having a preponderating incidence. Both may persist for weeks, months or years and to state that one is acute and the other chronic is erroneous. Once the initial response has occurred, a change from a serous to a mucus ear, or vice versa, is relatively uncommon. Uncommon though it may be, the change from the serous to the mucus is the more frequent. There is a high protein content of the serous fluid with excessive coagulability when exposed to air. The mucus fluid has a low protein content. In either event, the fluid is sterile. Many investigators have tried but have failed to demonstrate the presence of any of the pathogenic respiratory bacteria.

One cannot stress too strongly the fact that the process is one which involves the entire middle ear and not the tympanic cavity alone. The middle ear is a continuous anatomical structure from the mouth of the Eustachian tube to the last cell in the tip of the mastoid and the condition under discussion involves the mucosa of the mastoid cells as well as the mucosa of the tympanic cavity. This fact accounts for the rapid reappearance of fluid after removal of some from the tympanic cavity by mechanical interference. X-rays of the mastoid will give suggestive evidence that this is so, and operation on the structure does reveal the presence of fluid in the cells. Jervey,[10] Lawson,[11] Cody,[12] Tobey and others have testified affirmatively in this regard.

HISTORY.

A typical history will usually tell of a partial loss of hearing in one or both ears following a cold, usually accompanied by a tinnitus and the symptom of autophony. If there is air as well as fluid in the tympanum the patient may complain of "bubbles" in the ear, or state simply that at times there is loss of hearing, at other times none, depending upon the position of the head and the consequent movement of fluid in the ear. If the tympanum is filled with fluid there will usually be a complaint that the ear and head on that side have a "blocked feeling" and that the hearing is poor in the affected ear. In a fair percentage, the story of a previous cold cannot be elicited. It is well to remember that the disease can be and often is bilateral.

DIAGNOSIS.

The diagnosis is usually made by inspection; sometimes has to be made by auscultation; and, occasionally, can be made only by assumption which can then be supported by a diagnostic paracentesis.

Examination in the inflammatory stage shows a dilatation of the blood vessels of a drum whose landmarks are still present. If a conductive hearing loss is present which is somewhat out of proportion to the amount of pathology seen, it should make one suspect the presence of fluid. At a slightly later stage, the observation of a meniscus makes the diagnosis easy. Bubbles of air, too, when present in the fluid give the drum a characteristic appearance. If there is doubt about the existence of bubbles or a fluid level line, the use of the pneumatic otoscope can be of material diagnostic aid.

More difficult to diagnose is the ear, the tympanic cavity of which is completely filled with fluid and the only identifying feature is the overall amber color of the drum. The amber color can vary from a faint, barely perceptible, yellowish tinge to a dark, almost bluish cast approximately the color seen in the hemotympanum, yet even in the latter the amber tint is perceptible. This is the picture most frequently missed diagnostically. A helpful aid is the fact that in almost all of these drums the short process and the handle of the malleus stand out as chalky white in appearance, in contrast to the colored drum. This chalky white appearance of the short process and the handle of the malleus gives one the impression that the drum is retracted when, as a matter of fact, it may not be at all. The use of the pneumatic otoscope often shows up a wrinkled appearance to the drum, a finding not frequently found otherwise. In a majority of the ears which contain fluid of long standing, the drum, with the use of this instrument, will be found to be flaccid. In many of these, the drum will have a translucent appearance and look as though it were made of oiled paper. The picture, depicted above, variable as it may be from a light to a dark colored drum and thus somewhat confusing until readily recognized, represents the full tympanum (and probably the mastoid as well), and should be found 10 times as frequently as the drum with a meniscus. Until one reaches this diagnostic percentage more practice is needed in the examination of these ears.

Auscultation can be of help but is not needed for diagnosis if the amber color is recognized. The auscultatory sound is necessary for the diagnosis of fluid in the ear if an opaque drum is present and otitis media with effusion does occur in ears, the drums of which are opaque. The diagnosis of fluid in the ear is far more easily overlooked in such ears, for the loss of hearing present is usually assigned to the thickened drum (with possible adhesions).

There are a variety of auscultatory sounds. If there is some air as well as fluid in the tympanic cavity there is a resulting bubbling which denotes the presence of fluid. One never hears an auscultatory bubbling unless the tympanum has air as well as fluid in it. If the tympanum is completely filled with fluid, entirely different auscultatory effects are heard. The most usual is one which is like the chug of fluid heard when a large bottle partly filled with a heavy fluid is shaken. Even this description is inadequate. It is a difficult sound to recognize and interpret. Often it is heard only on the first auscultatory blow and subsequent blows will produce no sounds suggestive of fluid. Unless one listens carefully for the first blow, the diagnosis may be missed entirely. Occasionally one will hear a faint crackling immediately after the termination of the blow. Also, occasionally one hears only a roughened auscultatory sound, with no suggestion of fluid in it and the comparison with the auscultation sound in the well ear is needed to appreciate that there is something unusual in the affected ear. The necessity for making these discriminations is the reason why careful, gentle catheterization is needed, for the sounds which must be heard are clearly discernible only when very gentle inflation is practiced. It can easily be missed by a loud or forceful blow. The crude blow of politzeration, with its attendant extraneous sounds, may miss the desired one entirely. Infrequently, though certainly, one hears no sound at all and one wonders if the tube has been catheterized, but inspection of the drum in these instances will show bulging as compared to a precatheterization, normal contour.

The diagnosis of fluid in the ear can and should be aided by the employment of diagnostic paracentesis of the membrana tympanum. The use of paracentesis is certainly diagnostic and probably therapeutic as well. Its therapeutic employment will be mentioned later. The procedure is identical whether it is used for one or the other purpose.

Paracentesis can be done with a hypodermic syringe needle or a myringotomy knife. A majority of those who use paracentesis prefer the latter. In almost all instances a small opening (the width of the myringotomy blade) just anterior to the annulus at six o'clock will suffice. With a surgically clean myringotomy knife it can be done without anesthesia (even in children) and without sterilization of the canal, with no untoward results and with but little pain to the patient. It is wise to precede this with inflation. This will place the drum further from the inner tympanic wall than usual. The puncture will be less painful if the inner wall is not touched. If inflation has preceded the puncture, in most instances some of the fluid will flow into the external canal immediately following the removal of the knife from the drum. This will not represent all of the fluid in the tympanum and the removal of the remainder should be accomplished by further inflation or the use of suction. With inflation, the fluid, except in ears with a high mucus content, runs freely into the canal and out into the hollow of the concha. It has been called straw colored by many. No matter what name is given, it is this yellowish or amber colored fluid which gives the drum its characteristic appearance and whether the change in color of the drum in otitis media with effusion be light and barely perceptible, or dark and almost bluish, there will be the underlying amber to all except the opaque drums.

Suction with the use of the pneumatic otoscope (Siegle) which has been advocated following paracentesis and which creates a negative pressure seems to be contraindicated. Its use apparently aggravates the condition. This method does not remove as much secretion as does inflation or "spot" suction for much of the fluid drawn into the canal when the negative phase is used returns to the tympanic cavity when the negative pressure is released. The writer has tried this and with dismay has watched much of the fluid return through the paracentesis opening into the tympanic cavity when it was thought all of the fluid had been captured for the patient's relief as well as his. There are some who decry the use of inflation for the evacuation of fluid after paracentesis and urge only spot suction. The writer has employed both and can find no difference between them.

That there is a negative pressure in the ear at times can be learned by watching the drum following paracentesis. Very often a rising bubble can be seen as the negative pressure draws air into the tympanic cavity up through the fluid. Occasionally, following paracentesis, several bubbles of air can be seen doing this. On occasion, too, after a paracentesis and the removal of fluid by spot suction, one can see the drum change from its preparacentesis amber color to the usual, normal gray appearance, only to have the tympanum, seen by continued inspection of the drum, refill with fluid with a concomitant return of the amber color. This picture of a rising fluid level line is an intriguing one. The tympanum, under continued inspection and appropriate timing of spot suction, can refill as many as three times at one sitting. This is quite conclusive proof that the fluid is greater in amount than can possibly be contained in the tympanic cavity.

If the fluid in the tympanum is of the heavy mucus type, it may be necessary to employ additional measures in order to remove it from the ear. Often, in this type, it is necessary to make two openings in the drum, one an incision at the site of the usual paracentesis and another, a paracentesis, in the superior portion of the drum, preferably in the anterior portion. A heavy mucus secretion usually comes out in one mass.

On such occasions, unless the second opening is made by the time one-quarter, one-third or one-half of the mucus mass has been suctioned into the canal, the negative pressure in the tympanum is so great that the remainder cannot be suctioned from the cavity. On rare occasions it may be necessary to have an assistant inflate the ear while one is employing suction in order to remove all of the contents of the tympanum and give hearing relief to the patient. It is in this mucus type of otitis media with effusion that, with the employment of suction, one often sees a strand of mucus pulled two or three inches out through the paracentesis opening and into the canal by the suction tube. The color of the mucus has less of the amber tint than the serous fluid. Fortunately, these cases, which are more difficult to handle, are *in the minority. If, after paracentesis and removal of some* fluid, the patient reports the ear still feels dull or that the hearing remains dull, he is more than likely correct and one had better re-examine the ear and ascertain if there is not more fluid present. There well may be more fluid, particularly if it is of the heavy mucus type.

The paracentesis opening closes all too readily. In the great majority, if conscientious attempts have been made to remove *all the fluid possible, no further fluid will escape. Occasionally* some will continue to escape for a few hours and rarely the ear will drain for a day or so.

It may be argued that without sterilization of the canal, paracentesis may be dangerous on account of possible infection and subsequent suppuration. Having performed about 2,500 paracenteses, the writer has seen but one go on to suppuration and this was in a patient who had the very onset of a concomitant cold. The ear was dry and the hearing had returned to normal within a fortnight.

Apparently many paracenteses can be performed without subsequent injury to hearing. Hunt[11] has reported he has done over 80 in one ear, that of a doctor, with eventual cure and normal hearing. While few, if any, have made as many paracenteses as this, there is abundant evidence, if properly done, there is no real damage to hearing.

There is no characteristic hearing curve, for the pathology under consideration may and often does occur in ears already diseased. Pre-existing inner ear disease may also be present. In an uncomplicated ear, the presence of otitis media with effusion will, of course, give the picture of a conductive deafness. Usually the deafness is out of proportion to the pathology seen on examining the ear, though, on occasion, the fluid, small in amount, is in such position that it does not block the conductive pathways and the hearing is quite good. This, of course, is infrequent because in the great majority of these ears the tympanum is entirely filled with fluid.

It should not be forgotten that the diagnostic endeavors should be directed not only to the ear but to the discovery of general conditions which are abnormal.

PROGNOSIS.

It has been stated previously that fluid in the ear may remain for only a single day or it may persist for years. It is rarely possible to predict with certainty which ear may be fortunate and which may not. In general, it can be said that the ears with the greater amount of mucus content will present more of a therapeutic problem than the others. It must be remembered, however, that a serous fluid can persist in the ear for years. The smaller the amount of fluid in the ear the more likely there will follow a rapid recovery. If fluid has been present in the ear for a lengthy period before treatment is instituted, a protracted course is much more likely than if its presence is of short duration. Undiscovered, or untreated, complicating factors may result in persistence of the fluid. Repeated attacks, which occur with a moderate degree of frequency, suggest unrelieved local or general pathology. If the condition can be cleared, the hearing in almost every instance returns to its former level. Unless subsequent attacks produce adverse conditions, the ear apparently is as good, functionally, as before. On the other hand, if the pathological state persists, eventually changes take place which are detrimental to function and these are irreversible.

TREATMENT.

The treatment for otitis media with effusion is not specific and consequently challenging. In the early stages, therapeutic endeavors should be directed toward elimination of a respiratory infection if one is present. When the infection is over, treatment can be directed to the ear. Without antecedent respiratory infection, the ear should receive immediate therapeutic attention. Politzeration or catheterization may be employed once, or a few times, and in many instances this is all the treatment required. If the condition does not clear with these manipulations, attention should be directed to local and general pathologies or irregularities. At the same time, or later, according to the inclination of the attending otolaryngologist, therapeutic paracentesis with subsequent removal of fluid by politzeration, catheterization or spot suction may be employed. Direction of attention to local or general complicating factors may call for tonsillectomy or adenoidectomy, removal of secondary adenoid growth, correction of nasal abnormalities, X-radiation for lymphatic hypertrophy or regional lymphatic blockage (employment of radium applicators to the nasopharynx in particular has not been demonstrated to be of worth; Jordan has noted this), correction of dental malocclusion, allergic management, endocrine therapy, treatment for cardiac insufficiency, etc.

Thereapeutic paracentesis is decried by some, praised by others. The writer employs the procedure, even with the knowledge that it is not all sufficient because of the overwhelming relief that it affords the patient, for which the latter is so grateful he is willing to continue treatment even though it be protracted; because the majority require only one paracentesis for recovery and because the longer the fluid is present in the ear before treatment is instituted the longer the time required to effect a cure. Therapeutic paracenteses are carried out at varying intervals, according to the symptomatology of the patient, coincidental with other measures, local and general. It is considered that the employment of both is wise, as the use of one without the other is not so effective in affording a cure. As a last resort when all other measures have failed, a very small number may require mastoidectomy before relief can be obtained. If it is employed it would seem wise that the modern technique of a "closed" postoperative wound would be abandoned and the employment of an acrylic or tantalum tube extending from the antrum to the skin be substituted. The tube should be left *in situ* with the use of general biochemical coverage to avoid infection until evidence is present that fluid is not re-forming and, with "closure," there will be no return of it. A very few ears, despite many forms of therapy, will have a persistence of fluid. It is probable that these ears represent undiscovered complicating factors.

COMMENT.

This presentation is written with the hope that it may stimulate an increased interest in a wider recognition of otitis media with effusion. The mere printing of these words will not accomplish this. Introduced more than four months later to an otolaryngologist who listened to the verbal presentation of this subject at the last Academy meeting, the writer interestingly asked, "Have you diagnosed any cases?" The honest answer was, "I'm afraid I have not." That there would not be one case in an average otolaryngologic practice during a four months' period is incredible. No, words will not suffice. If ever this condition is to be widely recognized, it must be accomplished by personal demonstrations of the actual presence of fluid in the ear, particularly the ear with the full tympanum. This, in turn, must be done by those who have been privileged to have known teachers who have demonstrated it to them.

The challenge of otitis media with effusion remains. More effective teaching of this subject is required by those in a position to do so.

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indices. Vital capacities were also done with a modified spirometer; chest measurements were made at the level of the fourth interspace before and after arytenoidectomy. All studies were done under standard hospital conditions and at rest. Postoperative studies were made at intervals ranging from 24 to 36 weeks.

RESULTS.

Fig. 1 is the preoperative photograph of the patient in Case 1, a 33-year-old-female on whom a thyroidectomy was done 14 months prior to this study. Immediately after thyroidectomy she noted the inability to talk above a whisper, and following ambulation she noted increasing dyspnea upon exertion and orthopnea at rest. This was associated with palpitation and a marked inspiratory stridor. These photographs depict the utilization of most accessory muscles of respiration, both during deep inspiration as well as during quiet inspiration.

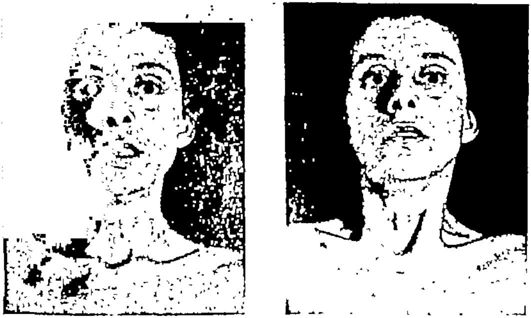

Fig. 1. Case 1, 33 years old, showing prearytenoidectomy respiration. (A) Quiet respiration. (B) Forced inspiration.

Fig. 2 represents the prearytenoidectomy spirometric tracings in the same case. There were predominantly sighing respirations occurring at approximately every 40 seconds, with accompanying laryngeal stridor. The actual rate of breathing averaged 12 respirations per minute and the tidal volume approximately 500 cc. The complemental sighs were approximately 1,700 cc. Vital capacity averaged 2,700 cc.

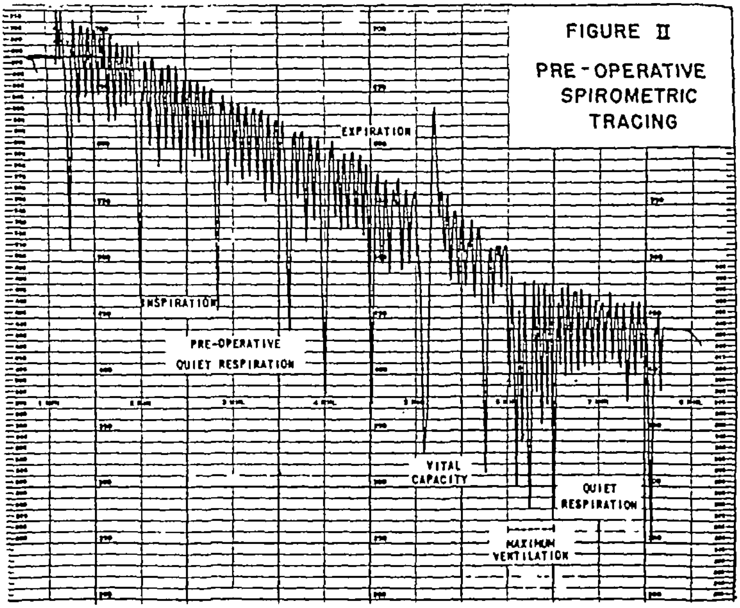

Fig. 2. Prearytenoidectomy spirometric tracings, Case 1. Note sighing respiration, slow respiratory rate, and low maximum ventilation.

The minute respiratory volume was 7,700 cc. The maximum ventilation per minute was 8,800 cc. (approximately 10 per cent of normal), and the maximum rate was 15.

In contrast, Fig. 3 represents the postarytenoidectomy photographs of the same case, and it is obvious that the quiet respiration as well as the deep respiration was improved. Both the intrinsic and extrinsic laryngeal muscles were very much enlarged at operation. The postoperative spirometry, as seen in Fig. 4, demonstrated the absence of deep, sighing respirations; and, although not demonstrated, the absence of stridor. More dramatic, however, was the increase in rate to the average of 18 per minute with a tidal volume of 400 cc. and a minute volume of 6,000 cc. Although there was little change in the vital capacity, the postoperative maximum respiratory capacity revealed an increase of approximately 31 per cent (8.8 to 12.7 liters).

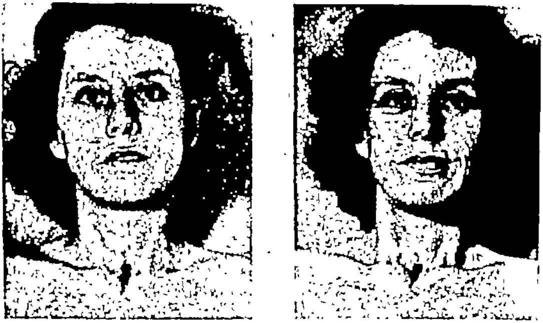

Fig. 3. Same case as Fig. 1. Postarytenoidectomy respiration. (A) Quiet respiration. (B) Forced inspiration.

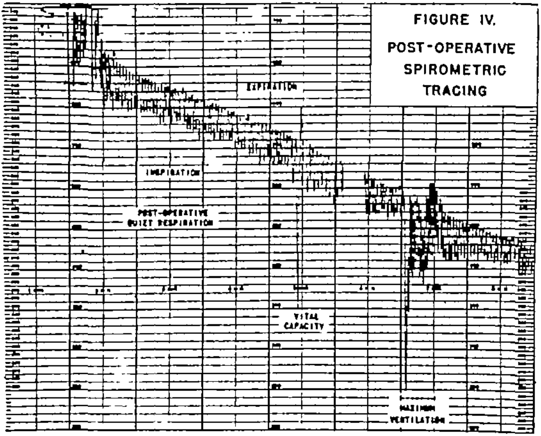

Fig. 4. Postarytenoidectomy spirometric tracings. Case 1. Note increased respiratory rate, absence of sighing and increased maximum ventilation.

Case 2 was a 56-year-old female whose thyroidectomy was done 12 months prior to study. Immediately following thyroidectomy there was a change of voice and dyspnea, palpitation and orthopnea were apparent on ambulation. Case 3 was a 65-year-old woman whose history of post-thyroidectomy bilateral palsy dated six years prior to her arytenoidectomy. Increasing dyspnea on exertion existed postoperatively with associated inspiratory stridor and palpitation. No spirometry was done on these cases.

It may be seen from Table 1 that in all cases there was some decrease from the theoretical normal in vital capacity and chest expansion prior to arytenoidectomy with an associated slow rate of breathing. In contrast, although there was little significant increase in vital capacity after operation, there were significant increases in both the chest expansion and respiratory rate. It might, therefore, be assumed that spirometric changes similar to those in Case 1 could be expected.

TABLE 1.

	Preoperative			Postoperative		
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3
Respiratory Rate (per min.)	12	13	14	18	16	16
Tidal Volume (cc.)	525			400		
Minute Volume (cc.)	7,700*			6,000		
Vital Capacity (cc.)	2,700	1,000	1,600	2,500	1,800	1,800
Maximum Respiratory Capacity (cc.)	8,800			12,700		
Chest Expansion (cm.)	2.54	1.27	4.44	5.72	3.81	5.72

*Including compensatory sighs.

Table 2 shows the result of the blood studies in all cases before and after arytenoidectomy. In both Cases 2 and 3 there was an increase in hemoglobin, total red count and hematocrat. In Case 1 there was a relatively normal hemoglobin and red count, although the hematocrit was slightly

TABLE 3.

	Preoperative			Postoperative		
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3
Hemoglobin (grams)	13.8	16.4	16.4	13.0	13.8	13.6
R. B. C. (millions per cm.)	4.24	5.44	4.71	4.54	4.87	5.9
Hematocrit (per cent)	47.0	50.5	51.0	46.0	45.0	47.0
M. C. V. (cubic microns)	112	98	106	101	98.7	80
M. C. H. (mm. grams)	33.0	30.5	34.8	29.0	28.7	23.0
B. P.	110/80	130/80	184/90	126/86	138/88	150/90
Pulse	100	90	80	100	80	70

elevated. In all cases the mean corpuscular volume was increased preoperatively, whereas the mean corpuscular volume and the mean corpuscular hemoglobin decreased. There was little change in the blood pressure, although there was some decrease in the resting pulse rate. Symptomatically, all patients showed definite and marked improvement in physical endurance. Their voices improved, stridor ceased and all were able to sleep without extra pillows.

DISCUSSION.

From these studies, it becomes apparent that arytenoidectomy performed by the method of Kelly and Woodman is a logical procedure for relieving bilateral abductor laryngeal palsy; furthermore, marked interference with the exchange of air is improved, as evidenced by the improvement in maximal respiratory exchange as well as by the improvement in thoracic expansion and respiratory rate. That compensatory mechanisms for the maintenance of normal tissue respiration are utilized is reflected by the relative increase noted in the preoperative blood cell indices. Postoperatively, these studies indicate that there is a tendency for the secondary or compensatory blood changes to return to normal values; furthermore, it is suggested that a means through which more oxygen is transported to the body is by relative increase in cell volume as well as hemoglobin concentration and not entirely through increase in the total number of red cells.

SUMMARY AND CONCLUSIONS.

From these preliminary and comparative respiratory studies of three cases representing post-thyroidectomy bilateral laryngeal abductor palsy treated by arytenoidectomy, it is apparent that:

1. The marked interference of the exchange of air accounts for the symptoms and evidences of chronic hypoxia.
2. The procedure is a practical means of correcting the interference in ventilation and preserving adequate voice.
3. After operation there is a definite return toward normal of the respiratory exchange of air as evidenced by the increase in respiratory rate, elimination of sighing respirations and stridor, the increase in maximum respiratory capacity; furthermore, a return towards normal of the red cell indices show this same trend.
4. It is suggested that in older patients the increase in actual cell size and hemoglobin is a compensatory mechanism which aids normal oxygen transport to the tissues (Cases 2 and 3).
5. It appears that in younger and more vigorous individuals the principal compensatory mechanism is an increase in ventilation (Case 1).

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NEW PILLING STORAGE BATTERY FOR E.N.T. INSTRUMENT LIGHTING.

Apparently the battery troubles of bronchoscopists and other physicians using battery-lighted instruments are over. George P. Pilling & Son Co., of Philadelphia, Pa., one of the country's oldest and largest surgical instrument houses, has just announced a new, portable, practically foolproof storage battery unit that is both powerful and dependable. What's more, it weighs only 16 pounds and can be easily carried around by even the smallest O.R. nurse.

The unit contains two 4 volt, 6 ampere nonspill storage cells in transparent plastic cases. Only one is used at a time, leaving the second always in reserve. A rheostat allows instant regulation of the brightness of the lamps, and a quick change from one battery to other by the flip of a switch does not require any changes of the instrument light cord tips. The condition of each battery may be determined by a glance at the charge indicator. And recharging is simple with the built-in charger. Just plug in the regular house current if it is the usual 110-115 volt, 60 cycle A.C. Descriptive literature is available.

HEAD AND NECK PAINS OF CERVICAL DISC ORIGIN.*

E. C. SCHULTZ, M.D., and R. E. SEMMES, M.D.
(by invitation),
Memphis, Tenn.

Headache is the most common human affliction and for centuries has clinically been associated with intracranial pathology. Most textbooks treat it solely as a symptom manifesting disturbed cerebral physiology, yet for almost 200 years the cervical origin of headaches has been suggested. Wolfe, in his excellent monograph, "Headaches and Other Head Pain," devotes less than a page to the neck as a source of headache. Other monographs often omit discussion of headache of cervical origin or attribute it to myositis, arthritis, spondylitis, tumors, Potts' disease and similar lesions. An abundance of evidence has accumulated so that we should consider the neck as one of the primary sources of head pain. Almost every neurosurgeon and many otorhinolaryngologists have had patients experience frontal, temporal or facial pain when the occipital region or neck was injected with local anesthetics. In almost all head injuries, trauma to the neck is incurred, and we now believe many of the post-traumatic headaches are due to cervical injuries. It is noteworthy that post-traumatic headaches are much less frequent following penetrating gunshot wounds than after blunt injuries to the head where the possibility of neck injury is greater.

One of us (R. E. S.) has observed cervical discs for many years and has always been impressed by the frequency of associated headache and facial pain manifested by these sub-

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*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 16, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

jects. Such pains are often the *earliest manifestation of* cervical disc pathology before the development of radicular symptoms occurs. In reviewing our own patients with cervical disc syndromes, we have found headache to be a prominent symptom in more than 50 per cent of those not requiring surgery. Among patients with severe pain in the upper extremity requiring surgery, less than 25 per cent list headache as a symptom.

Symptoms: Headache of cervical origin presents a rather typical clinical picture. The pain is chronic, recurrent and usually begins *in the suboccipital region on either side of the* head. After a few hours, the pain radiates anteriorly to the homolateral eye, forehead, temporal and occasionally maxillary regions. Usually described as a "dull ache or nagging pain," it may at times be throbbing in nature. This is especially true when the pain is retro-orbital. A common complaint is "it feels as if my eye is being pushed out." Such pain may last for several hours to several days. Rarely, patients have had constant pain for weeks when they are first seen. At times the pain is lancinating in character. Beginning in one suboccipital region, it may last for minutes or *hours, only to disappear, leaving a residual soreness of the* involved areas. Several such episodes may occur in a day, or days may intervene between attacks. At times pain may occur in only one region of the head or face, excluding all others. Thus, patients with only severe frontal, orbital, maxillary or temporal pain are frequently encountered. Occasionally, the homolateral eye, when painful, will water. We have never observed watering of the nose in this syndrome.

Examination of such patients may show remarkably little, especially if it is early in the course of the disease. Upon *close questioning, one is able to obtain a history of neck* injury, recurrent stiff or painful neck, or shoulder and upper extremity pain in a high percentage. Tenderness is usually present over the supra- and infraorbital nerves when pain in the frontal and maxillary region has been prominent. Likewise, *pressure over the greater and lesser occipital nerves in* the suboccipital region produces *extreme* pain. The brachial plexus of the homolateral side may also be extremely tender on palpation. When, on pressure over the occipital nerves, the brachial plexus or head (to compress the cervical spine) results in duplication of the patient's pain, we feel this finding to be highly suggestive, if not pathognomonic, of cervical disc disease.

Such individuals usually have a straightening of the cervical spine, the normal lordotic curve being lost. The posterior cervical muscles are spastic and often tender. Without realizing their neck is the source of trouble, some patients keep their head tilted slightly to one side, having observed that quick or excessive motion of the head results in severe head or face pain. That position of the head is often an important factor is exemplified by a recent patient who stated that his pain came on only following dinner. It was his custom to sit in a deep chair to read the evening newspaper. His position was one of acute flexion of the neck and, after a short period of time, this resulted in acute frontal and orbital pain lasting for hours and requiring opiates for relief. It is noteworthy that, although manipulation of the head may produce head or facial pain, traction on the head for a few moments does not give the same measure of relief it does in shoulder pain due to cervical disc ruptures. Likewise, pressure over the cervical spinous processes is less apt to cause head and face pain than shoulder pain.

In the diagnostic study of patients with chronic, recurrent headaches, X-rays of both the skull and cervical pain should be obtained. In the absence of clinical findings indicative of intracranial pathology, X-rays of the skull are of little help. Cervical spine studies reveal in early cases merely loss of normal cervical lordosis, while in more advanced cases intervertebral space narrowing, reversal of the cervical curve, hypertrophic lipping and ligamentous calcification are encountered.

Mechanism of Pain Production: The mechanism by which headache or facial pain due to lesions of the cervical discs are produced is unknown. Spasm of the posterior cervical muscles with resultant traction on the frontalis muscle has been used as explanation for pain of the forehead and orbit. Since tenderness of the supraorbital, infraorbital and occipital nerves is often present, a neurogenic source of the pain is suggested. Anatomically, the dorsal horn of the upper cervical nerves (*i.e.*, the sensory portion of these nerves), termed the substantia gelatinosa of Rolandi, emerges imperceptibly with the spinal tract of the Vth cranial nerve. Thus, theoretically, noxious impulses entering the upper cervical nerves may be referred over the distribution of the trigeminal or upper cervical nerves. Pain of lancinating character is more readily understandable by this theory. Further credence is lent the neural origin by the complete relief of pain often afforded these patients when very small amounts of 1 per cent novocaine solution are infiltrated around the occipital, supra- or infraorbital nerves.

Differential Diagnosis: The presence of chronic, recurrent frontal or maxillary pain leads many of these patients to seek the help of an otorhinolaryngologist. Indeed, many of our patients are referred to us by them when the sinuses are found to be free of disease. The chronicity of the pain, absence of infection, and normal appearing Roentgenograms, among other findings, readily excludes the sinuses as the source of pain. Trigeminal tic douloureux is often suggested when the upper two-thirds of the face is the painful area. True trigeminal neuralgia can usually be excluded by a careful history and always by witnessing an attack of pain. The pain of tic douloureux is lightning-like, momentary and excruciating. Several such episodes may recur in rapid succession. So-called trigger areas which, when stimulated, reproduce typical attacks are present. Cervical disc pain referred to the face is usually of many hours' duration and trigger areas are never present. The term atypical facial neuralgia is often applied to pain of this nature.

Migraine and histamine cephalgia are probably the two most frequent diagnoses encountered in patients with cervical disc lesions. The term migraine is far too frequently used to explain unilateral head pain. In a strict sense, it should be reserved for those individuals with recurrent, throbbing headache, usually unilateral at onset, accompanied by nausea, vomiting and irritability at the height of the attack and preceded by visual phenomena such as blurred vision, scintillating scotomata, photophia or hemanopia. Less frequently, speech disturbances, paresthesias and vasomotor disorders are encountered. A family history of migraine is usually obtained. Ergotamine or Dihydroergotamine 45 parenterally affords prompt relief of migrainous headache. The term histamine cephalgia on the other hand should be restricted to chronic, recurrent unilateral pain over the distribution of the branches of the internal and external carotid arteries, accompanied by vasomotor disturbances such as profuse watering and congestion of the eye, rhinorrhea, nasal obstruction, increased perspiration and frequently flushing of the skin. Such an attack may be precipitated by injection of 0.35 mg. of histamine base. Intravenous epinephrine in 1:400,000 dilution promptly terminates an attack.

Treatment: Since we believe the facial and head pain occurring in cervical disc lesions results from nerve root irritation, treatment is largely directed toward mechanically diminishing or preventing nerve compression. In the milder cases, merely cautioning patients against rapid or exaggerated head motions, the omission of a pillow during sleep and the use of heat in the form of a hot water bottle beneath the neck is all that is necessary. Among those with severe headaches, cervical traction affords satisfactory relief. Our routine is to hospitalize these patients for several days to acquaint them with the proper use of the traction apparatus. Five pounds of traction is applied with the usual cervical halter. Except for mealtimes and bathroom privileges, the traction is constant until the acute pain subsides. Gradually they spend more time out of traction when they are discharged to their homes and instructed to use traction only at night. The average hospital stay is about one week. Local anesthesia may be employed with considerable benefit in patients with severe headache associated with occipital nerve tenderness and in acute facial pain over the distribution of the supra- and infraorbital nerves associated with exquisite tenderness of these structures. A few cubic centimeters of

1 per cent novocaine infiltrated in the region of the occipital, supraorbital or infraorbital nerves often produces prompt relief of discomfort for hours. Longer acting anesthetic agents such as 2 per cent metycaine prolongs the relief of pain. When a considerable quantity of procaine is used; its systemic effect may give temporary relief.

Case Report: The following is a representative case of headaches due cervical discogenetic disease. C. L. W., age 56, the father of two physicians, was seen on Nov. 11, 1949. One week previously, pain occurred in the left occiput and radiated to the left eye. It was throbbing in nature, and constant, lasting about six to eight hours. Occasionally sharp, stabbing pain occurred in the eye. Although opiates were administered, pain relief was incomplete. One or more such attacks occurred daily. During the past 48 hours he had had no relief of pain. On questioning, he admitted previous mild neck discomfort, frequent cracking sensations in the cervical region but denied any previous neck trauma. Neurological examination was negative except for the following: The head was tilted to the right, and the normal cervical lordosis was lost. The posterior cervical muscles were in spasm and neck mobility was reduced. Compression of the head increased his symptoms, while traction for a few moments diminished them. Pressure over C-5 and C-6 spines, as well as momentary compression of the neck veins, increased the headache. The blood pressure was 160/100. There were no reflex or sensory changes. Our Roentgenologist reported "examination of the cervical spine shows narrowed intervertebral spaces at the fifth and sixth levels with some proliferative spur formations along the anterior margins and in the anterior ligaments. Very short nub-like spurs project into the foramina at the same level."

He was placed in cervical traction, using five pounds of weight. In *12 hours the pain was markedly diminished and in 48 hours it was gone.* The duration of traction was reduced daily so that after one week he was using it only at night. Pantopaque myelography was carried out on the seventh hospital day. Spinal fluid dynamics were normal. The fluid contained no cells and 56 mg. per cent protein. Myelography revealed failure of the cervical nerve root sacs to fill on the left at C-5 and C-6 levels. He was discharged the following day, with instructions to use traction nightly at home. He has had no further recurrence of pain.

CONCLUSIONS.

1. Headache and facial pain are common manifestations of cervical disc pathology.

2. When the typical pain is reproduced by head, cervical spine or nerve trunk pressure, pathology of the cervical discs is almost certain to be the cause.

3. Simple mechanical methods are usually sufficient to afford prompt relief of pain.

4. Surgical relief of nerve root compression is rarely necessary.

PRACTICAL TESTING FOR PURE PHYSIOLOGICAL HEARING FUNCTION.[*]

JULIUS LEMPERT, M.D.,
New York, N. Y.

The objective of this presentation is not to report newly discovered hearing tests. I am convinced that otologists are not suffering from a shortage of such tests. I will instead report to you what I found to be the best way of employing the long existing and well established hearing tests for estimating the correct degree and type of true hearing function still existing in the individually deafened ear.

I say "true hearing function" because there seems to be some confusion between the meaning of testing for the hearing function of a deafened ear and the testing for the ability of a deafened person to interpret correctly what little he hears with a deafened ear. I find it necessary to call attention to this distinction in the objective of testing hearing because I strongly feel that only the determination of true hearing function should be the concern of the otologist.

Only by determining the type, degree and extent of pure hearing function existing in a given ear can the otologist place himself in a position to correctly advise and institute when possible the necessary available means for improving physiological hearing in a functionally impaired ear.

In the presence of a hearing loss which is found by the otologist to be functionally incorrectible, the testing of a thus deafened person's ability to hear *ad libitum* with his limited

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[*] Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., New York City, N. Y., Jan. 5, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

hearing function, as well as the instituting of all available measures for improving his *ad lib* hearing should be left to the educator of the deafened.

I believe that the otologist's function in examining the hard of hearing patient should be to determine:

1. *What* each deafened ear fails functionally to hear under normal circumstances;
2. *Why* the individually deafened ear fails functionally to hear what it should normally hear under normal circumstances;
3. *Whether* the individually deafened ear which fails functionally to hear what it should normally hear under normal circumstances can be made to hear all or most of what it failed to hear, with the aid of abnormally amplified sound in one way or another;
4. *Whether* the ear which is not able to hear as it should normally under normal circumstances but càn be made to hear more normally by abnormal amplification of sound is still physically in good enough condition so that it could lend itself to the improvement of hearing by time-proven available medical or surgical means, so that the use of amplified sound will not be necessary;
5. *Whether* the hearing loss is of progressive nature, making it mandatory for the otologist to stress the importance of instituting medical or surgical measures for the arrest of such progress whenever possible.

For too many years hearing was being tested mainly to determine whether or not a hearing loss existed. Since there was very little known about improving hearing, the importance of accurately determining the type of hearing loss in a given ear and measuring its exact degree and extent was, therefore, not so essential.

Today, some definitely time-proven ways and means of improving hearing in certain types of deafness already exist; also, there are promising investigations being conducted which may lead to the ability of improving hearing in hitherto considered incurably deafened ears. In view of these facts, the necessity of carefully and accurately determining the type, degree and extent of an existing hearing function loss before advising measures for its relief and for determining the exact status of true hearing function after such measures have been instituted has risen to great importance.

I have been employing what I found to be the least fallible technique for testing hearing. It enables me to correctly diagnose the type, degree and extent of an existing loss of physiological hearing function within the deafened ear.

Both a correctly elicited history of the hearing impairment and a carefully conducted physical examination of both ears, prior to the testing of hearing function, are very essential.

The history of the onset and progress of the impaired hearing is preferably elicited not from the patient alone, but also from one closely related to and in constant association with the patient. To obtain a correct history of deafness from a patient seeking advice requires great clinical experience with the various middle ear, inner ear and mixed forms of deafness.

An understanding of the psychological factors, which will often prompt a deafened person to give a history of his deafness which can be very misleading to the less experienced otologist, is also essential.

After having taken the history I carefully examine the auricle, external auditory canal, tympanic membrane and middle ear structures and cavity of both ears with good illumination and if necessary with magnification. The findings are noted and an attempt is made to correlate the physical findings with the history given. If the physical findings are such as to render the veracity of the history doubtful, an attempt is made to obtain a more accurate history so that the physical findings and history could appear to be more plausibly correlated.

Having completed the history and physical examination, the testing for pure unadulterated physiological hearing function is begun in the following order:

1. Audiometry for pure tone sound.
2. Tuning Fork Testing for pure tone sound.
3. Speech Testing for ability to hear spoken words.

Audiometry for pure tone sound when executed patiently, carefully and objectively with a properly maintained correctly calibrated audiometer, the performance of which is constantly checked and verified, is today one of the most scientific means of measuring the degree and extent of an impairment of physiological hearing function for both air-conducted and bone-conducted pure-tone sound.

I sincerely believe that for the good of otology, no otologist who treats the deafened, either medically or surgically, should ever personally take audiometric readings either before or after having initiated such treatment. All audiometry is best performed by a well trained assistant whose sole interest should be to aid the otologist in making the correct diagnosis and to guide him in the correct evaluation of the results following his treatment. In my entire career I have never personally taken an audiometric reading either before or after instituting treatment for deafness. To date I do not know how to use an audiometer.

By carefully studying the decibel level of hearing for pure air-borne and bone-conducted sound in all the low and high frequencies, including the decibel differential between the air-conducted and bone-conducted sound, a correct diagnosis of the existing type of hearing loss in a given ear can be readily reached.

Tuning Fork Testing for pure tone sound, I employ with the following two objectives in mind: Since surgical treatment for deafness is indicated only for middle ear deafness and since it is to the patient's advantage to operate upon the ear with the greater impairment in air-conducted hearing, I first try to determine in which of the two ears the hearing impairment for air-borne sound is worse. Immediately before striking, placing and firmly pressing the 512 frequency steel tuning fork against the median line of the skull or of the inferior maxilla with teeth tightly shut, I slowly, clearly and loudly tell the patient that I know as well as he knows in which of his two ears his hearing is better; however, what I would like him to do is to carefully observe and tell me in which of the two ears does he hear this particular tuning fork better, while disregarding the fact that the better of his two ears is known to him. By supplementing the Weber test with such careful explanation to the patient of what is expected of him to observe and report, and using the 512 fork instead of a lower frequency vibrating fork, I was able to make the following observations:

a. The 512 tuning fork is heard equally in both ears when the cochlear nerve function is about equally good in both ears and the hearing for the air-borne sound is equally good or equally bad in both ears.

b. The 512 tuning fork is equally not heard in both ears when the cochlear nerve function is equally bad in both ears and the hearing for air-borne sound is equal or not equal in both ears.

c. The 512 tuning fork is heard in one ear only when the cochlear nerve function in that ear is good and hardly any or no cochlear function exists in the other ear.

d. The 512 tuning fork is heard better in one ear when the cochlear nerve function is better in that ear, regardless whether the air-conduction hearing is equal or inequal in the two ears.

e. The 512 tuning fork is heard better in the ear with the poorer hearing for air-borne sound when the cochlear nerve function is equal in both ears.

My second objective of tuning fork testing for pure sound is primarily the determination of the approximate degree of unused good cochlear nerve function reserve still existing in an air-conduction deafened ear, which could be utilized advantageously by a surgically reconstructed and functionally improved *air-conduction mechanism.*

For the above stated purpose the use of the 512, 1024 and 2048 frequency steel tuning forks is most valuable.

I always do these tuning fork tests myself. I believe that these tests should always be done personally by the otologist whose opinion and advice the deafened patient is seeking. This is also my opportunity to check the audiometric reading taken by my assistant. When my findings with the tuning fork tests are not in agreement with my assistant's audiometric reading, I order that the audiometric reading be taken again; however, in doing so I always insist that the recheck of an audiometric reading is done by an assistant other than the one who made the original audiogram, and without having had or being given the opportunity of looking at the audiogram which is being questioned. An assistant whose audiometric reading is being questioned, when permitted to retest the same patient, will as a rule not come back with greatly revised findings since he is not likely to admit having been careless in the first instance.

In testing with tuning forks the hearing of deafened persons who are seeking relief by vestibular fenestration, the important thing is to determine not how long they claim to have heard a particular tuning fork by air or bone conduction but rather to determine accurately the moment when they actually ceased to hear the tuning fork, which they claim to have heard. The determination of auditory acuity cannot be left entirely to the subjective response of the patient since this often leads to an erroneous estimation of the hearing status.

The veracity of the voluntary subjective responses to tuning fork testings for pure-tone sound, when these tests are executed in the traditionally accepted and employed manner, is often questionable. Especially so since many deafened persons with little hope for regaining their hearing in their desire to be declared as suitable for surgical intervention will not hesitate to try their best to convince the examiner that they have good bone conduction hearing which they almost all know to be a "must" if such surgery is to be considered.

In the presence of this desperation psychology among many of the deafened, I have learned long ago not to rely entirely upon their voluntary subjective responses. I investigated and found involuntary emotional signs and involuntary objective responses which vary with the presence and absence of hearing. The following involuntary signs and responses when observed have been found to be infallible and, therefore, I consider them as indispensable to the testing of hearing with the steel tuning forks:

1. The delayed raising of their hand as indication of not hearing the pure-tone sound at all.

2. The spontaneous joyful grin and elated facial expression immediately upon contact of the temporal bone as an indication of hearing the sound.

3. The sudden change of the joyful grin to the sad, forlorn look as an indication of having suddenly lost the sound.

4. The deviation of the eyeballs in the direction of the formerly heard sound as the first indication of their search for the suddenly lost sound.

5. The turning of the head in the direction of the previously heard, but now lost, sound when failing to locate it by deviation of eyeballs.

6. The final shutting of their eyes to concentrate on finding the lost sound when everything else failed.

The obvious interpretation of these emotional changes of expression and the involuntary responses to tuning fork testing permits the correct determination and evaluation of their hearing function for both air-borne and bone-conducted pure-tone sound as well as the correct time relationship between the two.

In order correctly to observe and evaluate these involuntary responses to sound when testing the hearing with the steel tuning forks, I invariably insist that the patient keep his eyes open and looks straight at me. I ask him to raise his hand as soon as he picks up and hears the sound of the tuning fork and drop his hand as soon as he begins to doubt whether he still hears the sound. By watching constantly for his involuntary responses to the sound and comparing and checking them with the voluntary responses, I find it possible by employing the involuntary responses as a control for the patient's voluntary responses to arrive at the correct estimation of the exact relationship between the air-conduction hearing and the hearing by bone conduction which can indicate whether non-utilized cochlear nerve function reserve is still present and when present its degree can thus be fairly accurately judged.

For testing with steel tuning forks, the 512, 1024 and 2048 speech frequency forks are used. After striking the tuning fork with a rubberized hammer, it is held close to the external auditory canal of the ear to be tested, while the other ear is being masked, until it becomes evident from both the voluntary subjective and the involuntary objective responses that the fork is no longer heard by air-conduction. Without striking it once again, the fork is quickly switched to the back of the auricle and placed forcibly and held tightly against the skin covering the mastoid process. Thus the ear is forced to pick up the pure tone sound by bone-conduction at an intensity which can no longer be heard by air-conduction. By carefully observing and correlating the patient's voluntary and involuntary responses to the bone-conducted sound, and timing the duration of its perception in a given air-conduction deafened ear, we can thus more or less correctly measure the degree of good cochlear nerve function reserve which is not being utilized by the functionally impeded air-conduction mechanism.

SPEECH-TESTING FOR DETERMINING WHETHER THE HEARING DEFECT IS CORRECTABLE OR NOT.

To determine whether dormant cochlear nerve function reserve which is not being utilized by a functionally impeded middle ear exists, the presently employed methods of testing hearing for the spoken voice are often misleading. These tests are inadequate because they are conducted in the language most familiar to the patient. The responses of the patient to words or sentences in the language best known to him do not necessarily represent pure cochlear nerve function alone. In many instances the responses to this type of speech-testing represent and are the result of a mixture of part cochlear nerve function and his ability to *ad libitum* interpret that part of the spoken word which the cochlear nerve function failed to supply. Such *ad-lib* hearing will vary with the I. Q. of the patient. This type of speech-testing belongs to the educator of the irreparably deafened patient.

Although, as a result of my great experience in diagnosis and treatment of deafness, I am thoroughly convinced that the degree to which the ability of an ear to hear the spoken voice has been reduced or improved can be readily judged from carefully taken audiometric readings of both air- and bone-conducted pure-tone sound, I, nevertheless, feel that in mixed forms of deafness, when the prognosis may be doubtful, speech-testing can often be a determining factor and govern the advice to be given the patient; however, if speech-testing is to serve best both the otologist and the patient, then such testing must be conducted in a language completely unknown to the patient or, still better, in meaningless words. The degree of a deafened person's ability or inability to repeat words with no meaning, when spoken into an individual ear with the other ear masked, is always representative of the degree and measurement of pure cochlear nerve function present in an ear. When employing speech-testing for diagnosis and advice, the degree to which such hearing is improved with the aid of the old-fashioned ear trumpet in a given ear represents the minimum amount of improvement that can be expected following the fenestration operation.

The following recorded meaningless words, or any other list of meaningless words, can be used for such testing:

boomsat
speerisk
booldrop
spitbook
hissteet
trapad
sismump
rumgup
spissbice
setdug
subnoy
pirhum
itchfitch
waycake
jesoon
chicherin
chetwierg
prosoutswo
baraban
snyeg
panavod
zissquiz
askwig
troopgrace
thudpuff
icepass
cobbitch
peepreap
shovetug
chestboost
buckmast
jaggap
gibzinc
browhard
ceboo
winkshank
chetnietz
svietlo
poydyom
parachod
zavstrak

CONCLUSION.

Experience has taught me that by carefully conducted cooperative testing of the deafened with the participation, direction and supervision of an otologist with good clinical experience and judgment, the difference between a correctable and noncorrectable true physiological hearing defect is easily recognizable.

TRAUMATIC PARALYSIS OF THE VOCAL CORDS.*†

MERCER LYNCH, M.D.,
New Orleans, La.

Although traumatic injuries of the larynx represent a negligible percentage of all types of trauma, with current methods of high speed travel, more laryngeal injuries are seen now than formerly. Most of these occur when the larynx is struck on the steering wheel of an automobile, causing a crushing injury of the thyroid or cricoid cartilage. The trachea is less liable to injury than the cartilages of the larynx because of the cushion of the thyroid gland, but injuries to the trachea are also encountered. Laryngeal injuries do not, as a rule, produce paralysis of the recurrent nerve, but the distortion and scar resulting from laceration of the laryngeal mucosa cause distortions within the larynx which either produce fixation of the vocal cords or limit the motion to such an extent as to simulate paralysis of the cord.

One of the main complications of thyroid surgery is injury of the recurrent laryngeal nerve. The incidence of such injuries varies from 1.5 to 3 per cent of operations on the thyroid, and such accidents are ever present possibilities. Most cases of abductor paralysis are a result of thyroid surgery. Ever since the advent of thyroid surgery the correction of this complication has been a problem for the laryngologist.

The history as well as symptoms of abductor paralysis is typical in the majority of cases. Immediately following operation the patient is unable to speak except in a hoarse voice or whisper. Difficulty in respiration occurs only in patients in

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*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 17, 1950.

†From the Departments of Otolaryngology, Tulane University of Louisiana School of Medicine, and the Ochsner Clinic, New Orleans, La.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

whom there has been much postoperative reaction and swelling or hematoma. In these cases tracheotomy is performed early. In the majority, however, the voice gradually improves, and by the time the patient leaves the hospital the voice is much stronger, although not normal. There is gradual improvement of the voice and with this gradual increase in respiratory difficulty. This becomes more severe with any form of physical exertion and upon lying down, when there develops a respiratory crow which is disturbing to other members of the household as well as to the patient. As this distress becomes more pronounced, activity becomes limited, and in order to obtain any rest the patient must sleep sitting up or propped up on several pillows. It is because of this increasing dyspnea that the patient soon seeks medical aid. The development of severe dyspnea is a gradual process requiring from six to eight months following injury.

If it has been necessary to perform a tracheotomy at the time of thyroid surgery or soon afterwards, it is advisable to wait six months before attempting restoration of the airway. This is to allow time for adjustment of thyroid patients, especially those with toxic disease. If, however, a tracheotomy has not been performed, then this should be done as soon as distress is noticed or when the patient is seen for the first time. This allows time for the patient to become adjusted to the tracheotomy tube and to overcome the immediate tracheitis and mucus associated with a recent tracheotomy. As soon as this period of adjustment has been accomplished, establishment of the airway may be attempted.

Since 1938, when Hoover[1] advocated submucous resection of the vocal cord in such cases, many surgical procedures have been suggested. Submucous resection provided an adequate airway in a high percentage of cases but resulted in a poor voice. Four years prior to Hoover's recommendation, Havens[2] had used a tracheotomy tube with a flap which enabled air to be exhaled through the cords but closed on inspiration. This provided a good speaking voice but made it necessary for the patient to wear a tracheotomy tube indefinitely.

It is, naturally, of primary importance to obtain an adequate airway without tracheotomy; a good voice, though desirable, is a secondary objective. To accomplish both, of course, would be ideal. With these objectives in mind, King,[3] in 1939, devised an operation in which the omohyoid muscle was used in an attempt to retract the cord for respiration and to establish some function of the cord for phonation. Kelly's[4] improvement of this technique consisted in arytenoidectomy via a window made through the thyroid cartilage. The latter procedure has resulted in a large number of successful results in his hands. Orton[5] modified Kelly's operation by removing the lateral half of the thyroid cartilage and then suturing the thyroarytenoideus muscle to the external perichondrium and the anterior split sternothyroid and thyrohyoid muscles. In 1946, Woodman[6] published his modification of the extralaryngeal approach; he advised a more open approach and a wider field for removal of the articular part of the arytenoid cartilage and fixation of the vocal cord laterally to the thyroid cartilage. Recently Thornell[7] proposed an intralaryngeal approach for removal of the arytenoid cartilage by means of suspension laryngoscopy. As this brief historical review shows, a variety of procedures and modifications of these has been attempted for relief of paralysis of the vocal cords.

I have had an opportunity to use several of the procedures mentioned in the preceding paragraphs. My experience with these will be briefly described. Like most other operations, these procedures have proved more successful in the hands of the originator than of those attempting to follow, until experience has made the pupil equal to the master.

The Kelly procedure, in which a window is made in the thyroid cartilage and the arytenoid cartilage is removed through this opening, was performed by me on three patients in 1945. The paralysis was the result of thyroidectomy for carcinoma of the thyroid in one patient; the result of the corrective operation was excellent in this case. The paralysis in the second case was produced by a traumatic injury to the larynx following a crushing blow to the thyroid cartilage;

the result of the Kelly operation in this patient was good. In the third patient the paralysis followed thyroidectomy for toxic goiter and the result was good, but the voice was poor, as both sides had to be operated upon in order to provide an adequate airway. The advantage of the Kelly procedure is that it enables the surgeon to grasp the cord at the vocal process and the pull is anterior and lateral, a fact which assures one of a more adequate airway. The second suture, placed at the middle of the cord, pulls directly laterally and this reinforces the first suture and enables one further to open the cord anteriorly, a feat which is difficult to perform by other techniques. The procedure has the disadvantage of requiring the surgeon to operate in an extremely small space. This makes dissection and removal of the arytenoid extremely difficult; furthermore, it is hard to maintain a dry field from bleeding muscles. These make the procedure tedious and rather long, but there is less reaction to the soft tissues of the larynx with resulting edema than in Woodman's procedure. I have since replaced Kelly's operation by the Woodman procedure.

The incision of the Woodman procedure is more extensive than that of the Kelly operation and gives a much wider field, better exposure and more adequate control of bleeding. I have observed that if the incision is made even more laterally than Woodman suggested, so that it is just at the anterior border of the sternocleidomastoid muscle, a more direct approach to the under surface of the thyroid cartilage is provided. One must be sure to disarticulate the cricothyroid articulation completely. Otherwise it is extremely difficult to rotate the thyroid cartilage for adequate exposure of the arytenoid. Also, more care has to be taken with this procedure to prevent tearing of the mucous membrane of the larynx. There is, moreover, a great deal more reaction locally and at the site of the arytenoid postoperatively than following the Kelly procedure; however, the advantage is that there is a great deal more space in which to work; the end-results have been satisfactory. Anterior lateral traction is obtained by hooking the suture around the lesser cornu and lateral traction by making a drill hole in the thyroid cartilage. Of the 12* patients operated upon to date, good results were obtained in all but one case. In that case the cord was overcorrected, and although the airway is excellent the voice is poor, but the patient is perfectly satisfied to be without the tracheotomy tube after five or six years.

The Thornell intralaryngeal operation is relatively new; I have had experience in only four cases. The approach is much more direct than the extralaryngeal route, and the operating time is greatly reduced. Removal of the arytenoid is not difficult with adequate exposure and in young persons, especially women, a great advantage is the absence of an external scar; however, the end-results have not been so good as with the external approach in my hands. In two of the cases an adequate airway and a good voice were obtained. In the first case the poor result was due to too enthusiastic use of the diathermy, which resulted in cicatricial contraction of the interarytenoid space as well as the lateral area. This patient required plastic surgery with eventual skin grafting before an adequate airway was established. The final results have been extremely satisfactory with a good voice. In the fourth case the airway has not been sufficient for full activity.

It has been my experience that in the internal approach the voice is as good or better than in the external approach, but the airway is not so good; therefore, as the primary objective is to provide adequate breathing space and, secondarily, a good speaking voice, I prefer the external approach and specifically the Woodman technique.

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VASOMOTOR RHINITIS.*

THEODORE E. WALSH, M.D.,
St. Louis, Mo.

That there is still no unanimity of opinion regarding either the etiology or the treatment of vasomotor rhinitis is evidenced by the abundant literature. In the cumulative quarterly index for the years 1944 to 1948, there appear no fewer than 87 articles under the heading, "Vasomotor Rhinitis." From the majority of these one is left with the impressions that *a.* vasomotor rhinitis is due to a hypersensitivity to some foreign protein, be it inhalant or food; *b.* antihistaminics will relieve its symptoms and *c.* that specific desensitization or dust or histamine therapy will cure the condition. Experience, my experience at least, contradicts these impressions.

Vasomotor rhinitis should be thought of not as a disease of the nose but as a manifestation of a systemic disease, the result of which is a sympathetic-parasympathetic imbalance resulting in peripheral vasodilatation, edema and secretion.

There is ample evidence to support the concept that a sympathetic-parasympathetic imbalance is the mechanism causing vasomotor rhinitis. E. P. Fowler, Jr.,[1] reported a case of unilateral vasomotor rhinitis following the resection of a superior cervical ganglion. The results of blocking the sphenopalatine ganglion are well known clinically. I[2] reported some 90 cases in 1932. Experimental evidence is presented by Blier[3] in which he extirpated the sphenopalatine ganglion in dogs and obtained marked vasoconstriction in the nose on the side of the extirpated ganglion. He also showed that the

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*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn. Jan. 17, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

parasympathetic fibres were the only ones with cell stations in the sphenopalatine ganglion. Corroborative evidence is afforded by the effects of the sympathomimetic drugs in the control of vasomotor rhinitis.

What then are the factors leading to such an imbalance? They can be stated as: *1*. Allergy. *2*. Endocrine dysfunction. *3*. Dietary inadequacy. *4*. Psychosomatic factors and a combination of any of these.

Allergy: I do not propose to elaborate on the allergic factors at this time except to say with the possible exception of true seasonal cases of allergic rhinitis, I find that allergy is not a common cause of the vasomotor rhinitis that we see repeatedly in the office. Bacterial allergy is perhaps one exception to this. There is considerable evidence that chronically infected membranes in the sinuses may give rise to an allergic rhinitis in which the bacteria in the membrane are themselves the responsible sensitizing proteins.

Endocrine Factors: I have neither the time nor the ability to discuss the mechanism of endocrine control of the autonomic nervous system. I would, however, like to mention cases illustrative of endocrine dysfunction resulting in vasomotor rhinitis.

Thyroid: Attention has been called to the part that thyroid plays in nasal disturbances by Proetz,[4] Laub[5] and others. I was first intrigued by this in 1932 when an Italian girl of about 20 years of age presented herself at the clinic for tonsillectomy. Her reason for wishing the operation was that her voice had become abnormal. It had assumed a nasal quality and was somewhat hoarse. She was an attractive girl who seemed, however, somewhat dull and apathetic. She had dry hair, dry skin and no vivacity. Examination showed her tonsils to be small and neither red nor obviously infected. She gave no history of sore throats. The chief finding on the examination was a typically "vasomotor" nose. The turbinates were purplish, boggy and swollen. There was some, though not excessive, secretion. Her mother, who accompanied her, stated that the girl was constantly tired, that she would retire to bed at 8:00 P.M., and it was with great difficulty that she was aroused to go to work the following morning. She was sent for a basal metabolic test and the B.M.R. was reported as minus eight. In discussing this case with the endocrinologists, they assured me that we could, under no circumstances, consider this a hypothyroid patient. They advised strongly against the use of thyroid extract in this case. I did, however, put the girl on increasing doses of thyroid and after about five weeks, when she was taking three and a half grains a day, she came back for examination with what appeared to be a perfectly normal nose, and her mother at that time stated that she was an entirely changed individual. She no longer needed much sleep. She had many dates and was out at parties and dances constantly. Indeed the girl had changed considerably in her personality. In the place of a rather phlegmatic, lifeless looking individual, there appeared a girl very bright and full of the joy of living. Another similar interesting case presented in a boy, M. B., 12 years of age, who was brought to me because, as his mother stated, "he has a cold all the time." This boy was fat, pale and without much vivacity. His mother told me that after school he would come home and sit and read a book and that he never wanted to go out and play with the other boys. His nose presented the typical appearance of vasomotor rhinitis, the rather pale, boggy turbinates with a fair amount, in this case, of clear, watery mucus. His basal metabolic rate was minus 19. We started him on thyroid extract and gradually increased the dose. At one time he was taking as much as 3½ gr. a day. His basal metabolic rate under this regime remained at minus 20. He was carried on with thyroid therapy, 2 gr., and for the past five years has had no colds nor any respiratory difficulty. After he had been on thyroid for awhile, his mother told me that he was a changed individual, that he now was full of energy, played with the other boys and indeed had been arrested on Halloween for some mischief which the gang had gotten into. He had, in other words, become a real boy.

We followed many of these cases of vasomotor rhinitis with low basal metabolic rates, and we have found that where a low basal metabolic rate is combined with a high blood cholesterol, the results from thyroid therapy are good. With a low metabolic rate and a normal blood cholesterol there are fewer good results. The least successful cases for thyroid therapy are those with a near normal basal metabolic rate and a normal blood cholesterol.

It is important in these cases to give sufficient thyroid to be of value. Our usual method of giving it is to start the patient with 1 gr. a day for perhaps two weeks and then increase to 2 gr. for two weeks to 3 gr. a day for two weeks and so on, being guided in the increase of the dose by the appearance of the nose. As a rule, about 3 to 4 gr. are necessary to cause a change in the nasal mucosa. When the desired change has been accomplished, we then reduce the dose until we find the optimum maintenance dose for the particular patient.

Ovarian Dysfunction: The relation of the nasal mucosa to ovarian function is well known and is evidenced by the changes observed during menstruation and pregnancy. We are all familiar with the occasional nasal blockage which occurs in the latter part of pregnancy which is immediately relieved when parturition has been accomplished. These same changes may occur as a chronic vasomotor rhinitis in some women at the menopause or in others who have been castrated. As an example, I would quote the case of Mrs. E. B. This woman of some 45 years of age was seen in 1941 with a bilateral chronic maxillary sinusitis, nasal polyps and much mucopus in both sides of the nose. She was diagnosed as having chronic maxillary sinusitis and had a bilateral Caldwell-Luc operation. Following the operation, when the nose had healed, the antrums could be washed and no pus obtained. The patient, however, constantly complained of a cold and when she was examined presented a red, rather dry and definitely swollen membrane. This is the type that Proetz[4] has described in his paper, "The Thyroid and the Nose." I was misled by the appearance and thought that this woman did have repeated respiratory infections. She was treated for these with various local treatments and with vaccine but with no success. After considerable time, in 1944, I was talking to her one day and trying to get some line on any general condition which might be contributary to her nasal state, and when I asked about her menstrual periods, she informed me that some years before she had had her uterus, tubes and ovaries removed and had not menstruated since. She was started on Diovocylin by injection once a week, and in three weeks her nasal condition cleared completely; she was followed thereafter by sublingual medication with Estrogen and for the next three years was seen at regular intervals without any complaints. She had no further respiratory infections and no further nasal trouble.

Testicular Hormone: Just as the nose reacts to abnormal function of the ovaries, so it reacts to abnormalities of testicular function. This has not been as publicized nor as widely observed as the condition in the female. Laub[5] reports a case of vasomotor rhinitis and polyps in a patient at the male climacteric. I would like to quote a case, I. B., a medical student, 24 years of age. This boy as a senior medical student came to the nose and throat service to do an elective. He had just recently been married and one day came to me complaining that he was deaf in both ears and had what he described as a very bad cold. The nose was completely blocked with swollen, wet membrane. There was much clear discharge. Both ears were completely full of fluid and the diagnosis was upper respiratory infection with serous otitis. The eardrums were opened and the fluid obtained, and on smears, this fluid was found to be loaded with eosinophiles. The diagnosis clearly was "allergy of the upper respiratory tract and ears." He was referred to the allergists who did the usual skin tests, finding no positives. Thinking, therefore, that he might be sensitive to something about his newly acquired wife, we hospitalized him and kept him isolated from his wife. Incidentally, this caused quite an upheaval in the family. After a month of this isolation, his condition was unchanged and he was no better. Under the general premise of his recent marriage and its attendant duties, I put him on testosterone therapy. Within six weeks, his ears had completely cleared, his hearing was normal and his nasal mucosa presented a normal appearance. He remained on testosterone therapy for some four or five months and since has been completely free of symptoms. I have known this boy well. He was in the Army and came back to specialize in otolaryngology and I am proud to have had him as a student.

Psychosomatic Factors: It is generally realized that emotional states may lead to peripheral vascular change. For example, the blushing that occurs with shame or embarrassment, the blanching that occurs with fear. I wonder, however, if otolaryngologists in general realize that emotional factors may play a large part in chronic organic changes, particularly in the nose. The experimental work which has recently been done at Cornell Medical School in this subject is particularly interesting. Work done in the department under the direction of Dr. George H. Wolff is well worth our attention. For example, in a paper entitled "Physiologic Mechanisms of Psychosomatic Phenomena," the authors indicate that changes may occur in the nasal mucosa and in the cellular response in the nasal mucosa from a psychiatric interview. They quote the case of one individual who suffered from what they termed chronic rhinitis. "A biopsy was taken from this person when he was in a period of complete rest and relaxation when membranes were in an average state of activity and again from the opposite turbinate at the height of a frustrating interview when the patient was on the verge of tears. Both biopsies were made with the same technique and the same topical cocaine anesthesia. The first section showed an essentially normal mucosal structure with moderate round celled infiltration. The second revealed the mucous glands to be filled with secretion and the vascular and lymphatic channels to be prominent and dilated. The cellular reaction of the nasal tissues was also greatly modified by symbolic stimuli. When the nasal secretions from the same subject were collected and stained by an appropriately standardized technique before, during, and after the discussion of significant conflicts, it was found that nasal hyperfunction was accompanied by marked eosinophilia, not only locally but in the peripheral blood as well." Consideration of this finding is extremely important to us when we have been told and we have believed in the past that in the presence of nasal eosinophilia, allergy,—and I refer now to a protein hypersensitivity,—was the sole cause. We should, I believe, revise our concept in this regard. Further experiments by this group of workers have demonstrated that, for example, when a known hay fever patient was put into a room in which there was a known pollen count, frequently there were no aggravated symptoms. A little sniffling, a little rhinorrhea, but no change in the color or swelling of the nasal mucosa. This same individual when situations about his personal life and interpersonal relationships were discussed had marked symptoms of hay fever with lacrimation and so on, until again at the end of the interview he was reassured and the nasal symptoms subsided.

DISCUSSION.

That the patient suffering from a chronically stuffy nose, postnasal drip, frequent colds, "sinus" and so on is, on the whole, unsatisfactorily handled is due, I believe, to the unfortunate misconception regarding the etiology of the condition. Such misconceptions are due, in large part, to the nomenclature of the disease. "Allergic" is the usual term applied. If by "Allergy" we mean "altered reaction," it is an admirable term. Common usage of the term "Allergy," however, has come to connote an antigen-antibody reaction, or a true protein hypersensitivity. This is, I think, unfortunate because immediately all attention is directed toward discovering the offending allergen and if this is not found, a nonspecific, desensitizing regime with dust extract or histamine is instituted. The other important factors I have mentioned above are consequently overlooked. For this reason, I think "Vasomotor Rhinitis" is a better term. By its use we may be led to think of other factors than allergy which influence the vasomotor function of the individual.

Again I would quarrel with the too easy acceptance of eosinophilia as a diagnostic finding in allergy, to the exclusion of other possibilities. The reports of Wolff and his co-workers are certainly worthy of further consideration. Eosinophiles are present in secretions and tissues in allergic rhinitis but their presence is not necessarily diagnostic of Allergy. They may be present in other conditions.

The fact that other causes of vasomotor imbalance, endocrine, dietary, psychosomatic, and so on are often overlooked may be due, perhaps, to specialism. A specialist must be constantly on guard against concentrating his attention on his particular part of the body at the expense of the patient as a whole. The otolaryngologist faced with a patient with vasomotor rhinitis is too apt to regard him as just a nasal or a sinus problem and forget that the patient has glands, a gastrointestinal tract, a brain, and for want of a better term, a soul. By these remarks, I do not mean to infer that we as otolaryngologists must also be endocrinologists or psychiatrists. I do insist that we should be able to recognize disease in other systems as causing symptoms referable to the ear, nose or throat and consequently seek the help of the appropriate specialist for further care. In these days it becomes a matter of team play between the otolaryngologists and other specialists if the patient is properly to be cared for.

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SURGICAL REMOVAL OF CONGENITAL NECK FISTULA.*†

COLBY HALL, M.D.,
Los Angeles, Calif.

Any discussion of congenital cysts and fistulae of the neck immediately involves two somewhat controversial problems: The first, etiology and classification; second, the rôle of the laryngologist.

Such efforts as the author has exerted toward the interpretation of branchiogenesis have ended in utter confusion and bewilderment. This has resulted, no doubt, from a combination of mental apathy toward the seeming fantasy of embryology and the impatience of an average clinician.

Dr. Charles Blassingame,[1] in 1947, presented a paper to the American Laryngological, Rhinological ond Otological Society, Inc., which stimulated a renewed interest in the subject.

While several etiologic theories regarding the origin of congenital lesions have been proposed, none has been universally accepted. "It seems more likely," according to Ward[2] in a recent publication, "that all the various theories have an element of truth in them." Meyer's interpretation of Wenglowski's work[3] gives us a clearer conception of the etiology and a more orderly classification of congenital pathology in the neck.

The terms branchial and branchiogenic have been used so generally and loosely that their true connotation is often disregarded.

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif., Jan. 21, 1950.

†From the Department of Otolaryngology, School of Medicine of the University of Southern California.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

Meyer arrived at certain conclusions which, if accepted, will help to organize our reasoning in this regard. These are (see Fig. 1):

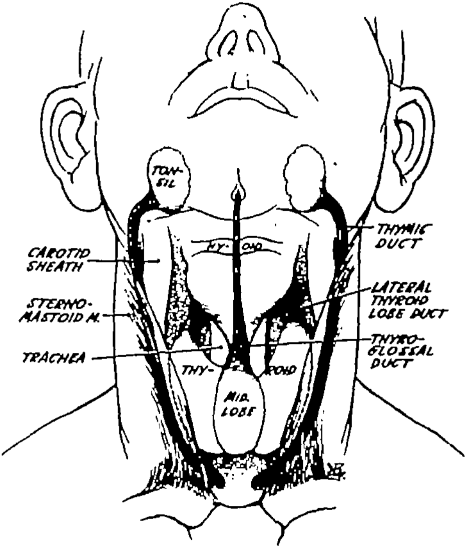

Fig. 1. Embryologic model projected upon adult contour for easier orientation (adapted from a model of Wenglowski).

1. The branchial apparatus disappears early in the second embryonic month. Only squamous rests, sometimes cartilaginous, can remain behind as parts of it. These are above and dorsal to the hyoid bone. Below the hyoid no rest of the branchial apparatus can remain behind. (Bailey and others have disputed this.)

2. The thymus develops from the third pharyngeal pouch. The thymic canal runs obliquely from the lateral pharyngeal wall to the sternum where the thymus gland develops.

3. The thymic canal retrogresses. It may persist throughout life or segments of it may persist.
4. The thymic canal rests may form a fistula or cyst. If the canal persists a complete fistula will result.
5. Lateral neck fistulae coincide with the thymic canal in direction and in histologic findings.
6. The lateral thyroid lobes have a short canal that disappears early in embryonic life. If a fistula or cyst resulted, the inner opening would be near the opening of the glottis. (If such exist, they are rare.)

He states definitely, "All congenital anomalies caused by the incomplete retrogression of the branchial apparatus must be located in the region above the lower border of the hyoid bone."

From these classifications we can make the following clinical classification of congenital cysts and fistulae (see Fig. 1).

1. Lateral cysts and fistulae which arise from the thymic tract.
2. Midline cysts and fistulae which arise from the thyroglossal tract and its ramifications.
3. Other cysts and fistulae which, if located at or above the hyoid, may be of branchial origin.

It would be presumptive, indeed, for the author to vouch for the academic accuracy of any embryologic research; however, from a clinical standpoint, the work of Meyer and Wenglowski gives us a concise classification and places the branchiogenic lesions in a limited anatomic area.

The treatment of these lesions is successful surgery. Successful surgery requires a knowledge of anatomy and entails the complete extirpation of the cyst or fistula.

The first case to be discussed was a complete lateral fistula which was totally removed with an excellent cosmetic result. This 19-year-old white boy presented a small fistulous opening about one inch above the right sternoclavicular articulation.

This had been present since birth and had intermittently discharged a sticky mucus. In recent years the frequency of this discharge had become a handicap to the patient. Lipiodol was instilled into the opening and radiographic studies revealed a tract upward to the pharyngeal region (see Fig. 2).

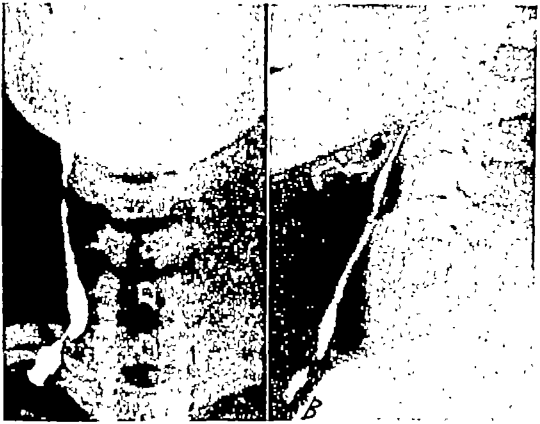

Fig. 2. Radiographic study of complete lateral fistula. (A) The tract follows the course of thymic duct.

Two weeks following a preliminary tonsillectomy the fistulous tract was removed surgically. A small filiform bougie which was passed through the tract greatly facilitated our surgical dissection. For cosmetic reasons the so-called "step-ladder" method was employed (see Fig. 3). Three transverse incisions were made, the first at the level of the fistulous opening. As the tract was dissected it was threaded from one incision upward to the next. Finally, when the thin wall of the pharynx was palpated, the tract was ligated, cut and removed from the neck wound (see Fig. 3). The three incisions were then closed. A very satisfactory result was obtained.

As Bailey[4] stated, "This step-ladder method leaves quite inconspicuous scars and robs dissection of a branchial fistula of most of its terrors" (see Fig. 3).

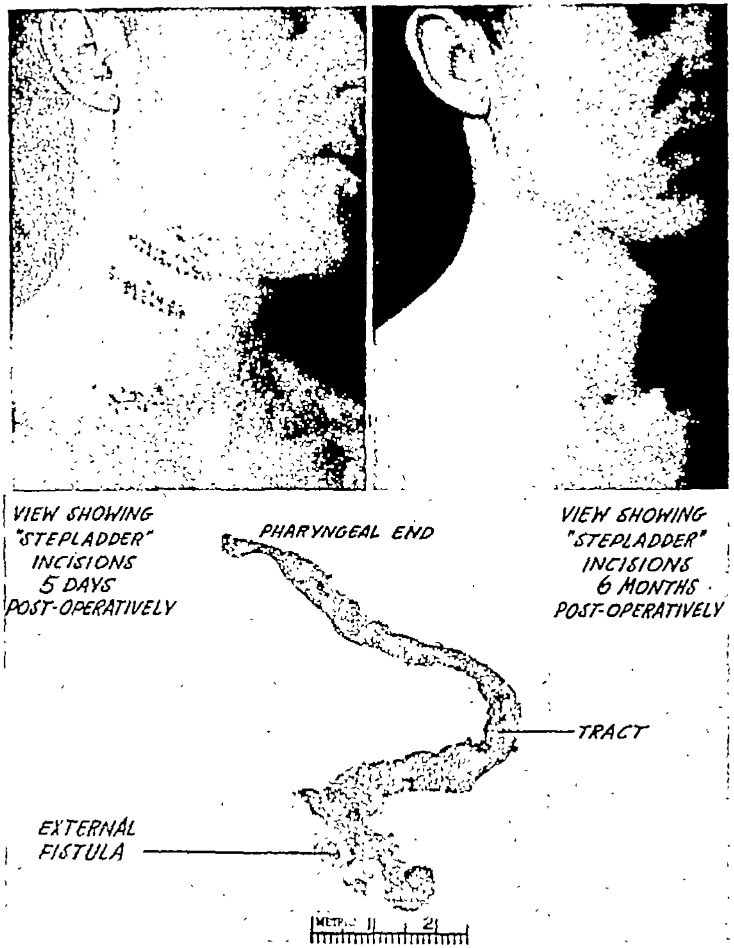
VIEW SHOWING "STEPLADDER" INCISIONS 5 DAYS POST-OPERATIVELY
PHARYNGEAL END
VIEW SHOWING "STEPLADDER" INCISIONS 6 MONTHS POST-OPERATIVELY
TRACT
EXTERNAL FISTULA
METRIC 1 2

Fig. 3. Photos showing excellent cosmetic result. The fistulous tract appears smaller than at surgery due to fixative agent used prior to the photograph.

Radiographically and anatomically, this tract followed precisely the same course as that of the thymic tract (see Figs. 1 and 2). From the tonsillar fossa it extended laterally and downward beneath the posterior belly of the digastric muscle. From here it passed medially and downward, anterior to the carotid sheath, to its opening along the anterior border of the sternomastoid muscle. This course of the thymic tract is said to be consistent. In one case we thought that an exception had been found because the opening was posterior to the sternomastoid, but the constant course of the thymic corridor was later proven when the tract revealed the fungi of actinomycosis.

The second case was a lateral cyst which was removed surgically. This 37-year-old white woman presented a large cystic mass in the right side of the neck just below the angle of the jaw (see Fig. 4). This mass had been present for eight

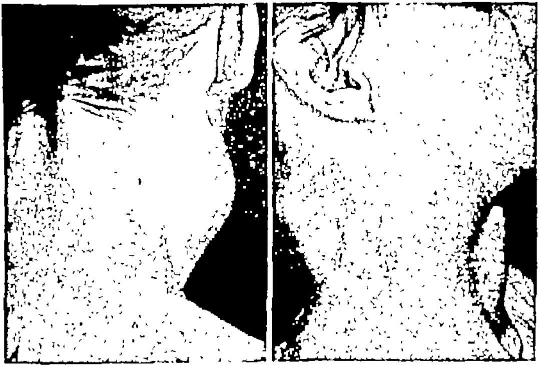

Fig. 4. Posterior and lateral views of lateral cyst. Small dark area in the lateral view resulted from the aspirating needle.

months. One ounce of opaque fluid was aspirated and lipiodol was instilled for radiographic study (see Fig. 5). The cyst was removed intact through a transverse incision. There was no duct or tract attached to it (see Fig. 6). It lay beneath the cervical fascia and upon the carotid sheath.

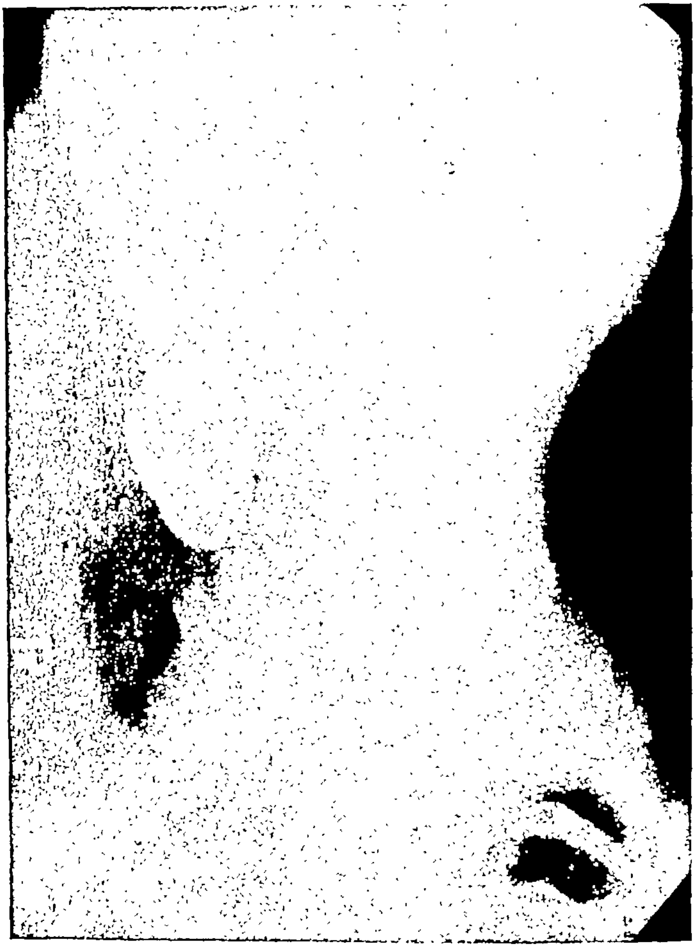

Fig. 5. Radiopaque study of lateral cyst.

As to whether this second congenital lesion should be considered of branchial origin seems academic. The important point is that it was characteristic of congenital cysts in this area. It appeared in adult life, was situated in the upper one-third of the neck and was filled with opaque fluid.

The question has been asked, "Are these lesions of the neck within the field of the laryngologist?" They are most assur-

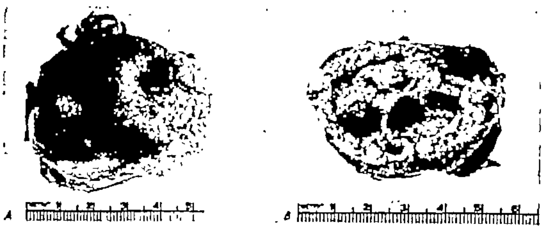

Fig. 6. Photos of specimen taken immediately postoperatively. (A) Intact specimen. (B) Inside of specimen after fluid was removed.

edly within the realm of the well-trained laryngologist. We should increase our efforts in the training of residents in this regard.

Certainly the pharyngeal end of a lateral fistula is in the field of laryngology, located as it is in the tonsillar area. With this pharyngeal attachment of lateral and thyroglossal tracts already in our favor, our interest and teaching should be such as to retain all congenital neck lesions in the field of laryngology. This can be attained only by the thorough training of residents. The diagnosis and surgical anatomy of congenital cysts and fistulae should be included in the programs of all teaching residencies in otolaryngology.

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1136 West Sixth Street.

CYTOLOGICAL SMEAR TECHNIQUE IN THE DIAGNOSIS OF CARCINOMA OF THE MAXILLARY SINUS:

PRELIMINARY REPORT.*

G. S. FITZ-HUGH, M.D.;† C. N. MOON, JR., M.D.,†
and C. H. LUPTON, JR., M.D.‡,
Charlottesville, Va.

The cytologic method of cancer diagnosis has been attracting increasing interest since the publication of the monograph by Papanicolaou and Traut[1] in 1943. Papanicolaou and his associates in the study of exfoliative cells of the female genital tract developed a technique in the preparation and staining of vaginal smears so that neoplastic cells could be recognized. From these initial studies there have evolved methods for examining the cellular elements of the secretions and contents of other body areas,[2] such as the lung,[3] stomach,[4] urinary tract[5] and nasopharynx.[6]

We have applied the cytologic smear technique to the study of washings from the antra in an effort to evaluate its usefulness in making the difficult early diagnosis of carcinoma of this sinus. All too often the diagnosis of antral malignancy is not made until gross clinical and radiologic evidence of such involvement is manifest. Should an acceptable degree of certainty be demonstrated by the smear technique, then the relatively simple procedure of antral lavage could be more fully utilized as a diagnostic aid.

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*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 17, 1950.

†Department of Otolaryngology — ‡Department of Pathology, School of Medicine, University of Virginia.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

The study of the cells derived from antral washings requires that the cytologist be familiar with the cells from noncancerous disease of the antrum such as suppurative, allergic, or other varieties of sinusitis.

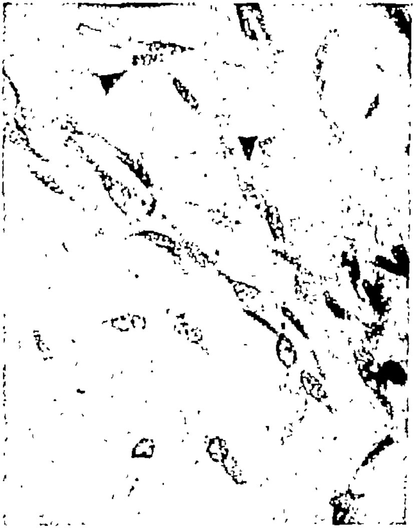

Fig. 1. Antral washings showing normal ciliated columnar epithelium. 680×.

In this preliminary report we have limited our cases to those in which malignancy could with reason be suspected; *viz.*, those presenting evidence of disease in a single antrum. Washings from 72 patients were studied, among which six cases of carcinoma were found. Unfortunately, all six of these cases exhibited cancer in advanced form which could have been recognized readily by other methods of diagnosis; however, since we were able to distinguish the tumor cells so readily in antral washings from these six cases, we are hopeful that earlier forms of the disease can be recognized with an equal degree of accuracy.

Technique: The majority of the antral irrigations were performed under topical cocaine or pontocaine anesthesia. Several lavages in children were done while under ether anesthesia. The antra were entered with a curved, blunt cannula through the middle meatus, or with a straight needle through the inferior meatus, depending upon which approach was thought to be most desirable in the individual case. Warm normal saline was used as the irrigating solution. Approximately 50 cc. of the washings were collected and to this an equal quantity of 95 per cent ethyl alcohol was added. The combined solutions were centrifuged as soon as possible for 30 minutes at medium speed (3000 r.p.m.). The sediment was collected and spread evenly on slides previously coated with egg albumin. The slides were then immersed in a solution of

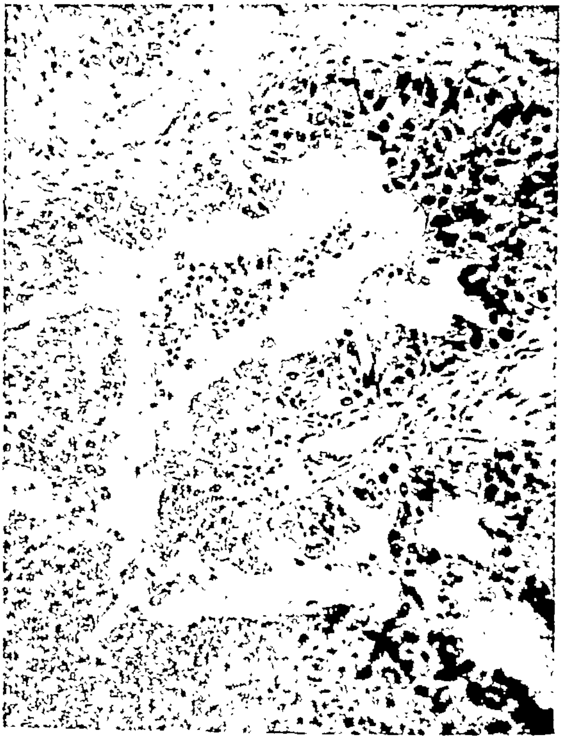

Fig. 2. Carcinoma of antrum showing exfoliation of tumor cells. 150X.

equal parts of 95 per cent ethyl alcohol and ethyl ether. The staining technique followed essentially that devised by Papanicolaou,[1] using hematoxylin, orange G-6 and E-A 65. The completed slides were mounted with cover slips and Canada balsam or permount.

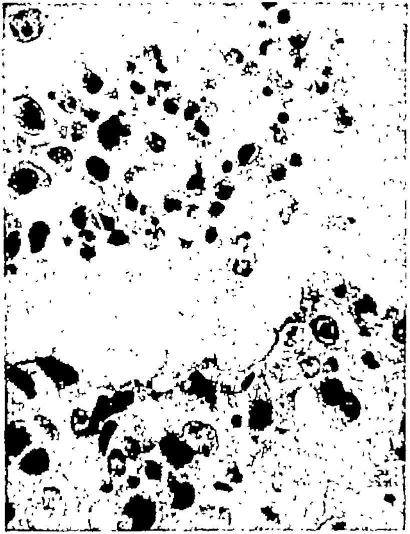

Fig. 3. Higher power magnification of Fig. 2. 550×. Note resemblance of exfoliated cells to malignant cells in tissue.

Microscopic Findings in Noncancerous Antral Disease: The washings from 66 patients were examined. As one would expect, the smears of the washings encountered in this group of patients contained a wide variety of cells. A few erythrocytes were usually present and easily recognized. As all the patients had some type of sinusitis, leucocytes were nearly always present. The polymorphonuclear leucocytes and lymphocytes were readily identified. Eosinophiles and mononuclear phagocytes were sometimes seen. A few stratified squamous epithelial cells may be seen, but most of the epithelial cells were tall, columnar and ciliated in type. Numerous variations may be encountered, such as columnar cells with no cilia, as well as oval and round cells, the latter cells being

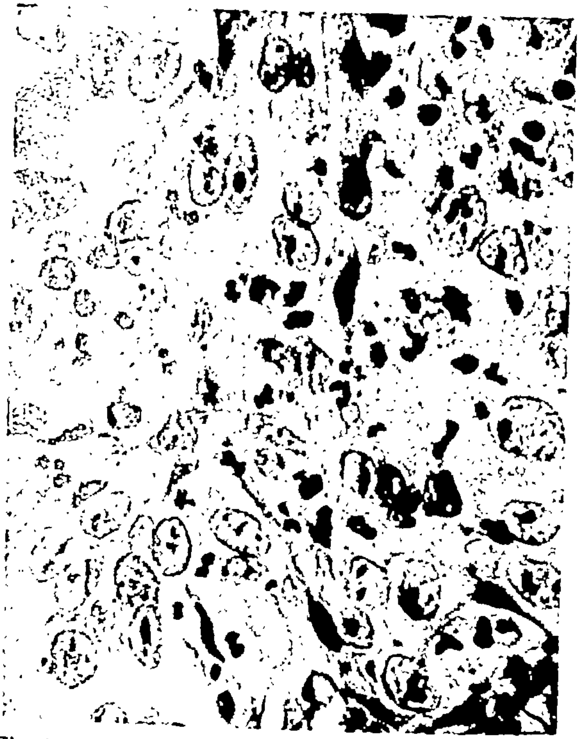

Fig. 4. Section of carcinoma of antrum (Case 1). 650×.

found most frequently with chronic suppurative sinusitis. The epithelial cells may appear singly or in small groups. Not infrequently degenerative cells were seen which could not be definitely classified. These may be epithelial or inflammatory in origin and at times may offer some confusion in diagnosis. Some of the cells encountered are demonstrated in the photomicrographs.

In reporting the findings from the washings the following classification as stated by Papanicolaou was used:

Class I: Smears that are negative, containing no atypical cells that may be confused with cancer cells.

Class II: Smears that are essentially negative but which contain atypical cells not generally confused with cancer cells.

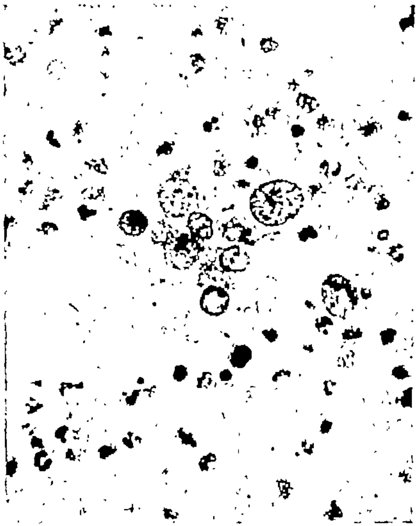

Fig. 5. Antral washings from Case 1 showing exfoliated tumor and inflammatory cells. 650×.

Class III: Equivocal smears, containing abnormal cells suggesting a cancer but insufficient in quantity and characteristics to justify a positive diagnosis of cancer.

Class IV: Smears containing individual cells and groups of cells with sufficient characteristics to identify them as cancer cells.

Class V: Smears similar to those in Class IV showing characteristics even more diagnostic of malignancy. In this series no diagnosis of Class V was made, due to lack of experience in interpreting these washings.

Microcopic Findings in Neoplastic Antral Disease: Over a period of 12 months we have had six patients with carcinoma

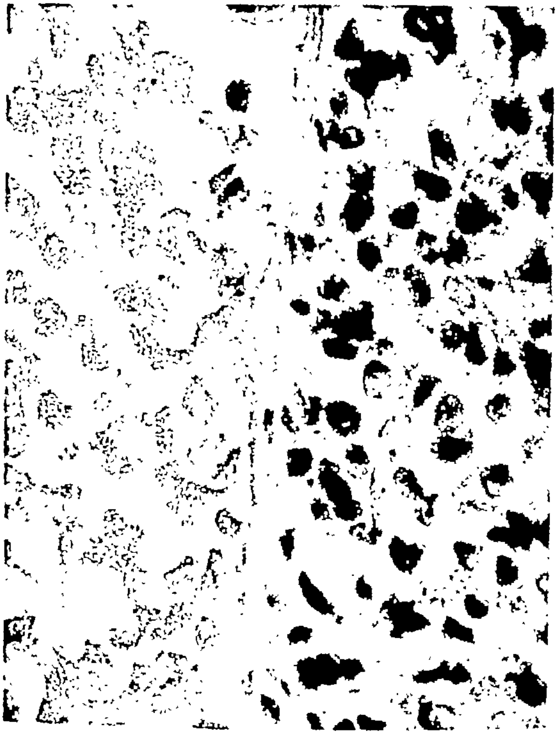

Fig. 6. Section of carcinoma of antrum (Case 5). 650×.

primarily of the antrum. There have been carcinomas of the nose, involving the antra secondarily, which have not been included in this group. The smears in the washings from these positive cases showed the same cellular elements as in the noncancerous group, with cancer cells in addition. Some of the cells are demonstrated in the accompanying photomicrographs. The cancer cells appear individually and in groups, with a fairly marked difference in the shape and size of the individual cells. The nuclei are large and abnormal, with prominent nucleoli and abnormal chromatin arrangement within them. Basophilic staining reactions in these nuclei are frequently encountered. Mitosis is not often seen. In the epidermoid types the cytoplasm may show varying degrees of keratinization manifested by orange color. The cytoplasm was unusually scanty when compared with that of the normal cell and may show vacuolization. It was mainly the abnormal characteristics of the nuclei of the suspicious cell or group of cells that led to a positive diagnosis of cancer.

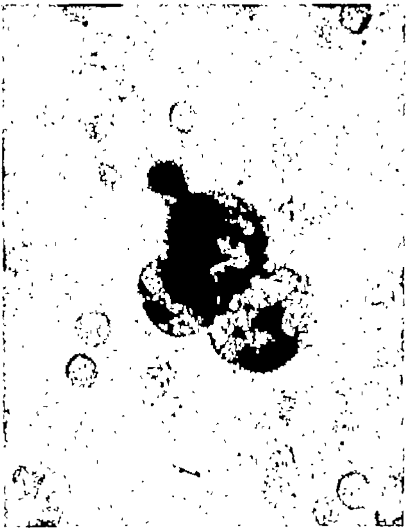

Fig. 7. Antral washings from Case 5 showing exfoliated tumor cells. 1100×.

Some details of these six cases, all proven by biopsy to have carcinoma of the antrum, may be seen in the accompanying table:

CLASS IV AND CLASS III PAPANICOLAOU STAINED SMEARS

	Patient	Sex	Age	Color	Class	Pathology Reports
1.	C.M.	F	42	W	IV	Epidermoid Carcinoma, grade II.
2.	H.C.	M	17	W	IV	Unclassified Malignant Tumor (Favor Endothelioma).
3.	H.J.	M	7	W	IV	Diffuse Endothelioma.
4.	J.G.	M	62	W	IV	Epidermoid Carcinoma and Adenocarcinoma, grade IV.
5.	D.H.	M	50	W	IV	Adenocarcinoma, grade III.
1.	C.P	F	21	C	III	Epidermoid Carcinoma, grade IV.
2.	V.M.	M	25	W	III	Chronic Maxillary Sinusitis.

Two Class III cases were encountered. One was shown eventually by biopsy to have carcinoma of the antrum and the other chronic sinusitis. To place these cases in a false negative or false positive category is open to some question, as Class III is a recognized equivocal group. Exploration of the antrum in patient No. 1 of the Class III series disclosed a carcinoma with intact and grossly normal mucous membrane over the tumor area (subsequent washings following preparation of this paper were reported as Class IV). Patient No. 2 in the Class III group was found to have chronic suppurative sinusitis upon exploration. This equivocal report was due most likely to cells exfoliated from an inflamed mucous membrane which had areas of metaplasia in its epithelium. It would appear that all Class III washings should be repeated in an effort to obtain a more satisfactory diagnosis.

Patient No. 4, after frequent washings over a six months' period, subsequent to X-ray therapy, became a Class II, then again returned to a Class IV with clinical evidence of recurrence of the carcinoma and positive tissue examination. This suggests that the Papanicolaou stained washings may be of aid in the follow-up of treated cases.

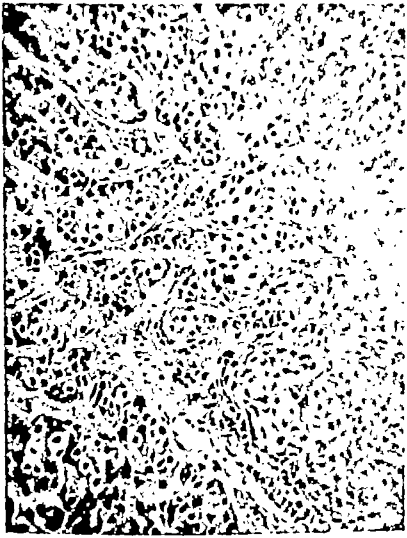

Fig. 8. Section of carcinoma of antrum (Case 4). 160×.

Patient No. 5 was interesting in that an initial Class IV smear became a Class I, following surgery and X-ray irradiation, and has remained so for over eight months. Tissue examination at that time was negative for recurrent carcinoma.

Another patient not included in this group with radiologic and clinical evidence highly suggestive of carcinoma of the antrum showed negative washings. Exploration disclosed a large dermoid cyst.

Comment: Among 72 consecutive cases of unilateral antral disease, six cases of carcinoma have been recognized without serious error by the application of the Papanicolaou technique to antral washings. The diagnosis of either sinusitis or carcinoma was later confirmed by the clinical behavior of the case or by biopsy.

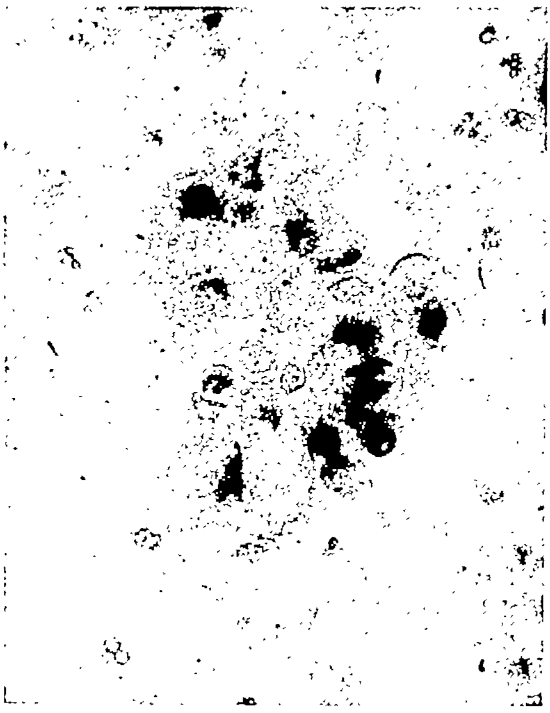

Fig. 9. Antral washings from Case 4 showing typical group of exfoliated cells. 690×.

It, therefore, appears that cytological studies of antral washings should be recognized as a useful aid in the diagnosis of carcinoma of this region.

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NEURINOMA OF THE PHARYNX.
REPORT OF A CASE.

W. G. KENNON, JR., M.D.,
Nashville, Tenn.

This type of tumor is known by many names. Neuroma, neurinoma, neurilemmoma, neurilemnoma, solitary neurofibroma, gliofibrosarcoma, Schwannoma, peripheral glioma and fibroma of nerves can be considered synonyms. These tumors originate from the fibres of peripheral nerves. The exact origin is probably the neurilemma or Schwann's sheath of nerve fibres. These tumors are characteristiaclly ovoid in shape, well encapsulated and firm. The cut surface presents a glistening, gelatinous appearance and is quite firm. Histologically, there are spindle shaped cells which have a tendency to arrange themselves in parallel formation, the so-called palisading effect. The nuclei are also spindle shaped, containing nuclear chromatin and reticulum. There are usually one or more nucleoli. Interspersed between cells and groups of cells are reticulum fibres and sometimes a considerable amount of collagen. It is thought by some investigators that the occurrence of a tumor of this type is but a solitary manifestation of Von Recklinghausen's disease. Indeed, Von Recklinghausen's disease has been observed in several patients having neurinomas of the head and neck.[1]

Figi, in 1933, reported such a tumor in the pharynx, and said that he could not find a similar one in the literature. Since that date, about 15 such tumors have been reported as occurring in the pharynx. Undoubtedly there are many unreported ones. These neoplasms are always benign, slow growing and never cause trouble until they are of such proportions that their size produces symptoms. The most common symp-

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Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 28, 1950.

toms are difficulty in swallowing and altered speech. On examination the tumor mass is easily seen projecting into the pharynx. In some cases there has been noted a Horner's syndrome on the same side.[2,3] Diagnosis is usually established by pathological report of tissue removed at the time of operation. In some instances aspiration biopsy has been successfully undertaken prior to operation. The treatment is surgical, and there are no reported recurrences. Some surgeons advocate the external route[6,7] with the preliminary precaution of ligating the external carotid. Those men reporting the intraoral approach experienced no difficulty in accomplishing complete removal of the tumor mass without excessive bleeding. Postoperative complications have been Horner's syndrome, dysphagia due to paralysis of the soft palate, unilateral laryngeal paralysis. One early postoperative death was reported.[9] This followed carotid ligation and the exact cause of death was undetermined. The paralyses observed postoperatively are to be expected when one removes a tumor having its origin in nerve tissue. These neoplasms are extremely radioresistant, and Roentgen therapy is never advocated.

CASE REPORT.

In September, 1949, a somewhat obese, 25-year-old, white, married female was seen for the chief complaint of difficulty in swallowing, and impaired speech. Her mother had noticed enlargement of the left tonsil in her daughter since the age of eight or 10. For the past five years her voice had been affected and had an adenoidal quality.

Examination revealed a large mass in the left pharyngeal wall which displaced the faucial tonsil medially as far as the midline. The appearance was in many respects similar to a large peritonsillar abscess, without the signs of inflammation, of course. Palpation revealed a firm, but not overly hard, mass. There was no palpable pulsation. The remainder of the examination of the ears, nose and throat revealed nothing abnormal. Under general anesthesia with ether the tonsil was peeled off the tumor mass with ease, revealing the tumor to be behind the pharyngeal musculature. The muscle layer was split and an attempt was made to remove the tumor in one piece. It was a well encapsulated, ovoid mass of large proportions and was so firmly attached posteriorly that I could not deliver it in one piece. It was removed piecemeal with a tonsil snare, care being taken to avoid the external carotid artery which was found to be displaced laterally by the mass. The tumor was easily removed in this manner, leaving a large cavity in the neck. Into this vacancy a finger could be passed superiorly to touch the transverse process of a vertebra high above the level of the soft palate. Inferiorly, the cavity extended well below the base of the tongue and the fingertip could be placed directly on the exposed and laterally displaced carotid sheath. There was minimal bleeding throughout the operation. Any approximation of the incised tissues was decided against, because of the possibility of inhibiting drainage, and the cavity was left open. Penicillin was given postoperatively, and convalescence was uneventful. Postoperatively, the patient experienced difficulty in swallowing and this has persisted to date. There is almost complete paralysis of the soft palate on the operated side. She manages solid food very well, but liquids have a tendency to return through the nasal passages. Her voice is normal.

PATHOLOGICAL REPORT.

Gross Pathology: The specimen consists of numerous varied sized masses of tumor tissue weighing 40 gm. and small amounts of attached fat and muscle. The tumor tissue is of a uniform moderately firm consistency and pale yellow in color.

Microscopic Pathology: The predominant histologic structure of the tumor consists of oval and fusiform nuclei lying in a palisade arrangement about rounded pink-staining areas free of nuclei. In smaller areas the tumor appears more collagenous without regimentation of cells. Mitoses are absent. This is considered to be a neurilemoma, type A.

SUMMARY.

1. Neurinomas are benign tumors having their origin in the fibres of peripheral nerves.

2. These tumors cause symptoms only by virtue of the size they sometimes attain.

3. Treatment is surgical removal; postoperative complications are paralyses of the nerves involved.

4. A case of neurinoma of the pharynx was presented.

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706 Church Street.

AN UNUSUALLY LARGE TRACHEAL FOREIGN BODY (FOUNTAIN PEN CAP) WITH CASE REPORT.

LINCOLN C. DICKEY, M.D.,
Cleveland, Ohio.

The literature is voluminous with reports of many varieties of foreign bodies in the food and air passages. It is extremely rare to encounter a tracheal foreign body as large as a fountain pen cap.

Jackson first called attention to the fact that each foreign body encountered presents its own problems. The following case report will further illustrate this point.

CASE REPORT.

T. T., a 12-year-old female, was brought to the emergency room at St. Luke's Hospital about 11:30 P.M. on Oct. 1, 1949, approximately one and one-half hours after she had accidentally choked on the cap of a fountain pen. She had been rushed to a neighborhood clinic where an X-ray demonstrated the fountain pen cap apparently in the trachea at the level of the bifurcation. Since she was not having any respiratory distress she was brought to St. Luke's Hospital where adequate bronchoscopic equipment was available.

Family and past history was noncontributory.

Physical examination was normal except for distant breath sounds over the left lung fields. There was no dyspnea or cyanosis.

The X-ray brought with the patient showed a fountain pen cap lodged at the level of the bifurcation of the trachea with the open end up. In order to more conclusively localize the foreign body, the patient was taken to X-ray for a lateral view of the chest. Within a few seconds after the film was taken, the patient suddenly began to struggle for air, became very dyspneic and cyanotic. She was taken to surgery where her color improved with the inhalation of oxygen.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 29, 1950.

A small amount of intravenous sodium pentothal was administered and as soon as relaxation of the jaw muscles was obtained, a 7 mm. Jackson bronchoscope was inserted directly, through the cords and into the trachea. The foreign body was found wedged into the opening of the left main stem bronchus with the open end tightly against the right lateral wall of the trachea above the bifurcation. The open edge of the cap was grasped with forward grasping forceps, freed from its lodging and brought up into the trachea. The open end of the cap had too great a diameter to protect it with the beveled end of the bronchoscope. The patient's condition necessitated immediate removal of the obstruction, so the cap had to be raked past the cords in order to remove it. The

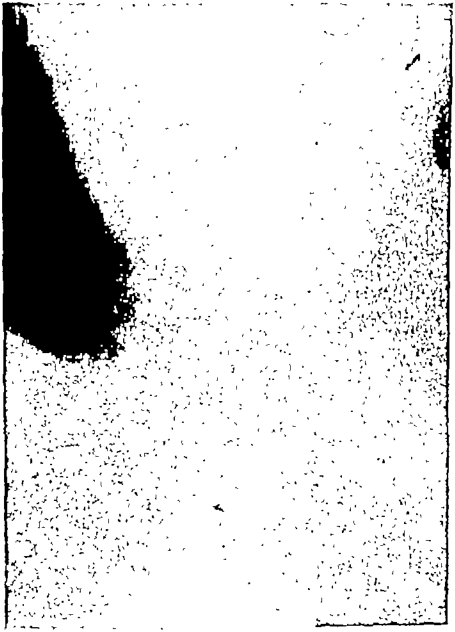

Fig. 1.

larynx was examined immediately afterwards. There was a small amount of bleeding in the region of the right cord and considerable congestion and beginning edema. It was deemed advisable to do an immediate tracheotomy. This was done under local 1 per cent procaine anesthesia. A No. 6 tube was inserted; the wound was partially closed with silk sutures to the skin, and the patient was returned to the room in good condition.

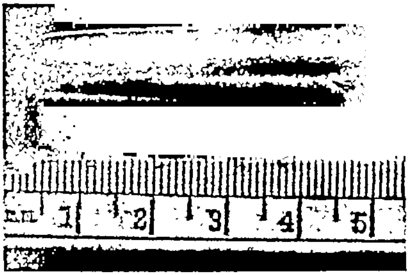

Fig. 2.

Eight hours later the patient could barely squeak any air through the larynx. The tube was blocked off on the fourth postoperative day and was removed on the fifth postoperative day. Secondary closure of the wound was done two days later.

Laryngoscopy one week following the procedure revealed a normal larynx. The voice was completely normal. The patient was discharged from the hospital on Oct. 10, 1949. Indirect laryngoscopy one month later revealed a normal larynx.

COMMENT.

There are two factors of special interest brought out by this case. First, it is surprising that so large a foreign body could lodge in the trachea without causing immediate signs of respiratory obstruction. The absence of a severe cough reflex is probably explained by the immobilization of the foreign body as it became wedged in place just above the bifurcation. The sudden onset of dyspnea and cyanosis can be explained. This occurred when the gradual swelling and edema, caused by the foreign body, suddenly reduced the amount of air which could bypass the foreign body.

The second point worthy of discussion is the method of choice in removing such a large foreign body. It seems logical that removal of the foreign body through a tracheotomy opening would certainly be the procedure of choice. This would avoid any possibility of laryngeal trauma; however, to use this method, it is necessary to do it before the patient's condition becomes so critical because of severe respiratory obstruction. Again we see the importance of time.

SUMMARY.

1. A case of an extremely large foreign body (metal fountain pen cap) of the trachea is presented.

2. Removal of the foreign body by bronchoscopy caused laryngeal trauma.

3. There may be a delay before onset of severe obstructive symptoms.

4. One should, therefore, consider the presence of a large foreign body in the trachea to be an immediate emergency.

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In Memoriam

RAOUL CAUSSE, M.D.,

1892-1949.

It is with great regret that we learned of the passing, on Nov. 16, 1949, of Dr. Raoul Caussé, Director of the Laboratory of Experimental Otology at l'Ecole des Hautes Etudes in Paris. In addition to holding this position, Dr. Caussé was a laureate member of the French Academy of Science. He was also the founder and, for many years, president of the French Society of Otolaryngology and editor of the *Annales d'Oto-Laryngologie.* He was consulting physician of the French Air Force and professor at l'Ecole nationale superieure des Telecommunications. It is unfortunate that Dr. Caussé's work was not better known by American otolaryngologists, physiologists and audiologists. He possessed the rare combination of facility in disciplined research and sensitivity to the needs of clinicians. His more than 200 publications include work on cochlear microphonics and action potentials, electroacoustics, the effects of noise on animals, postgalvanic nystagmus and the toxic action of streptomycin on the vestibular system. He developed many unique histological techniques and always insisted on histological controls in all of those experiments where it was possible.

It was my pleasure to visit Dr. Caussé in his laboratory last Spring. I was impressed by his versatility, not only as an intelligent clinician, but also as a physiologist and mathematician. He spoke of looking forward to a trip to the United States to acquaint himself with progress in otolaryngology, audiology and the neurophysiology of the auditory system.

Dr. Caussé is survived by his widow and daughter, both of whom are physicians. His son-in-law is also an otolaryngologist and it is hoped that he will continue the fine work of his father-in-law.

In the perspective of time Dr. Raoul Caussé will loom as a great figure in otolaryngology.

S. R. SILVERMAN.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

There will be a joint three-day meeting, Sept. 11, 12 and 13, 1950, of the North Carolina Society Eye, Ear, Nose and Throat and the South Carolina Society of Ophthalmology and Otolaryngology. Headquarters will be the Skyland Hotel, Hendersonville, N. C.

The following otolaryngologists will be on the program:

Dr. Fletcher D. Woodward, University of Virginia School of Medicine, Charlottesville, Va.

Dr. Lester A. Brown, Emory University School of Medicine, Atlanta, Ga.

Dr. B. T. Horton, Mayo Clinic, Rochester, Minn.

Dr. Donald F. Proctor, Johns Hopkins University School of Medicine, Baltimore, Md.

Ophthalmologists to be present are:

Dr. Bruce Fralick, University of Michigan School of Medicine, Ann Arbor, Mich.

Dr. Edmund B. Spaeth, University of Pennsylvania School of Medicine, Philadelphia, Pa.

Dr. Frank Walsh, Johns Hopkins University School of Medicine, Baltmore, Md.

Dr. Richard G. Scobee, Washington University School of Medicine, St. Louis, Mo.

For any further information, please contact: Dr. MacLean B. Leath, Secretary, North Carolina Society Eye, Ear, Nose and Throat, High Point, N. C., or Dr. Roderick Macdonald, Secretary, South Carolina Society of Ophthalmology and Otolaryngology, Rock Hill, S. C.

INTERNATIONAL COURSE IN AUDIOLOGY, STOCKHOLM, SEPTEMBER, 1950.

In a letter to the editor, published in Vol. 37, No. 6, of *Acta Oto-Laryngologica,* it was proposed that a practical course in audiology should be arranged in Stockholm this year.

In the editor's reply the desirability of arranging such a course was emphasized, and all those who might be interested were requested to communicate with *Acta Oto-Laryngologica.* The response was most encouraging, and the large number of inquiries and preliminary enrollments attest the very keen interest that has been aroused by the proposal. The signatories of this communication have therefore formed a committee for the purpose of organizing such a course, to run from the 11th to the 20th of September.

The following lectures will be given:

A. *Lectures:*

I. Anatomical, Physiological, Physical and Psychological Backgrounds.

II. Hearing Loss—its prophylaxis, incidence, causes and psychology.

III. Hearing Tests. *1.* Physical Basis of Audiometry. *2.* Comparison between old and modern methods. *3.* Tone Audiometry — basic and special tests — interpretation. *4.* Speech Audiometry—basic and special tests—interpretation. *5.* Group Audiometry—basic and special tests—interpretation. *6.* Standardization of Determinations, Audiometers and Audiograms.

IV. Treatment of Hearing Loss. *1.* Treatment of Hearing Loss in Children. *2.* Medical and Surgical Treatment of Hearing Loss, Except in Otosclerosis. *3.* Fenestration.

V. Remedies. *1.* Speech Re-education. *2.* Lip Reading. *3.* Hearing Aids. *4.* Rehabilitation and Re-employment. *5.* Social, Psychological and Educational Aspects. *6.* The Audiology Center.

B. Practical Courses and Demonstrations:

I. Tone and Speech Audiometry—Basic and Special Test Methods.

II. Selection of Hearing Aids.

III. Fenestration: training on specimens . (The course will be arranged if the amount of interest warrants it. Reservations can be made only for a few participants. A special fee will be payable to cover the costs of specimens, instruments, etc.).

The above survey is not to be regarded as the final program for the course. Broadly speaking, it is proposed to give the theoretical training in the forenoons, with two or three lectures for the entire assembly, and the practical training for two or three hours in the afternoons to small groups of participants.

For further information, address C. A. Tegnér, Secretary, 33 Birger Jarlsgatan, Stockholm C. Sweden.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1950.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 108BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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VOL. LX MAY, 1950. No. 5

THE PLACE OF THE OBLITERATIVE OPERATION IN FRONTAL SINUS SURGERY.*†‡

EDWARD H. LYMAN, M.D.,
St. Louis, Mo.

I. INTRODUCTION.

This paper is presented as a study of the problem of frontal sinus surgery. The literature is reviewed. Three illustrative cases are reported from a series of 17 cases of frontal sinus infection in which surgery was performed. In 11 cases an obliterative operation of the frontal sinuses was done. In two cases a gold Ingals' tube was permanently inserted. In four cases a modified Lynch type operation was done. One case from each of these three groups is reported in detail. An analysis of these cases is presented in an attempt to arrive at a rational basis for the proper type of surgical treatment of frontal sinus disease.

II. HISTORICAL REVIEW.

The literature on frontal sinus surgery is voluminous and controversial. Time and again the pendulum swings toward

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*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

†From the Department of Otolaryngology, Washington University School of Medicine.

‡Published with the approval of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration. The statements and conclusions published by the author are a result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 27, 1950.

and away from radical surgery. Ogston,[1] in 1884, was the first to report opening the frontal sinuses for the drainage of infection. Riedel,[2] in 1898, first described the complete obliterative operation on the frontal sinus. This apparently attracted little attention in this country. In 1903, Killian[3] described his operation for frontal sinus disease. In the classical Killian operation, the entire floor and all of the front wall of the frontal sinus, except a bony bridge to preserve the orbital ridge, are removed. The Killian operation attracted widespread attention and became very popular in this country.

In 1905, there were Symposiums on frontal sinus disease at the Annual Meetings of both the Americal Laryngological, Rhinological and Otological Society, Inc., and the American Laryngological Society. There were several other articles published that same year. There was great interest in surgery of the frontal sinus. Coffin[4] discussed various external operations which were being developed and classified them in three types: *1.* the Ogston-Luc with a relatively small opening into the sinus through the anterior wall, *2.* the Killian operation, *3.* the open method, attempting to obliterate the sinus by packing through an open wound (Kuhnt-Coakley). Although he performed radical operations on the frontal sinus, he decried "frenzied surgery." He also mentioned the desirability of X-rays which were just coming into use. Richards[5] reported 14 cases, most of which had a stormy course with several operations, usually starting with a Killian operation and ending up with external obliteration. Coakley[6] discussed frontal sinusitis, acute and chronic, and used a radical obliterating type of operation with packing of the open wound. In the acute cases he first infracted the middle turbinate or removed its anterior end. If improvement did not follow, he performed a radical operation promptly. The severe cases were desperate and frequently died. The idea of waiting until the acute symptoms had subsided before radical surgery was done had not been presented. In his chronic cases the mortality rate was much lower. At the same time other authors strongly urged reliance on intranasal treatment. Casselberry[7] was very emphatic, citing the high mortality and the great deformity of the radical operation. Ingals[8] first described his flanged, expanding gold tube, which is still in use today. As he originally presented it, it was for intranasal introduction with a special introducer and was left in place only temporarily.

The first wave of enthusiasm subsided as the dangers of frontal sinus operations became apparent. Logan Turner[9] discussed operative treatment of chronic frontal sinusitis. He said that there were two main principles. In the one, the sinus was preserved and, in the other, the sinus was obliterated. He felt that the Killian operation was the best procedure and gave a higher percentage of cures; however, he stressed the high operative mortality and cited 24 cases of death following operation. Freudenthal[10] also stressed the dangers of surgery. He felt that the majority of cases would improve with intranasal treatment, but that some would need radical operation. He made a large nasofrontal opening. It was not realized until much later that the resulting scar tissue had a strong tendency to contraction.

Barnhill[11] discussed the surgical treatment of empyema of the frontal sinus. He stated that it was necessary to remove completely all pyogenic structures, to provide ample drainage of the operated parts and to get the cavity to heal either by a healthy nonsuppurative lining or by complete obliteration of the space by means of granulation tissue. Of the types of operation he preferred the Coakley type of external obliteration but admitted the great deformity. He took note of the controversy between proponents of conservative and radical treatment. He also emphasized the dangers of both intranasal and external frontal operations. Knapp,[12] in 1908, was the first to approach the frontal sinus externally through the floor; however, if the sinus was large he made a small opening through the anterior wall to reach the upper part of the sinus. Knapp's operation never received widespread recognition until Lynch much later popularized the approach through the floor of the frontal sinus.

In England there was much disagreement about surgery of the frontal sinuses. Many men were dismayed at the deformity and high mortality rate; however, Sir St. Clair Thomson, Watson Williams and others[13] favored the radical operation, either a Killian type or an osteoplastic flap type. Watson Williams was one of the first to mention cicatricial stenosis of the nasofrontal duct after operative enlargement.

In 1912, MacKenty and Cocks[14] discussed the various radical frontal sinus operations. They classified them in three groups: *1.* removal of the anterior wall (Ogston-Luc, Kuhnt), *2.* removal of the floor (Jansen), *3.* removal of both the anterior wall and floor (Riedel, Killian). They preferred Knapp's method of going through the floor and reported six successful cases.

Dissatisfaction with frontal sinus operations has led to the presentation of many modifications of previous procedures. This has continued to the present time. In 1913, McKimmie[15] described a modification of the Killian operation. In the same year McKenzie[16] presented the first comprehensive article in English about diffuse osteomyelitis from nasal sinus suppuration. He said that the osteomyelitis was secondary to infection, either spontaneously or postoperatively, and was more apt to follow acute cases in both categories. In the cases of operated osteomyelitis recorded to that time all patients died. This was a very important step in the realization of operation in the acute phase of disease as a cause of osteomyelitis.

Imperatori[17] reported a case, in 1915, which developed meningitis from an exacerbation of a chronic frontal sinusitis four years after a Killian operation, and died. At autopsy the nasofrontal duct was found to be closed off. This is cited as an instance of the early recognition of the importance of patency of the nasofrontal duct in cases in which the frontal sinus is not completely obliterated. Beck[18] was one of the earlier authors to emphasize the cicatricial closure of the nasofrontal duct after any operation which destroys the mucous membrane and disturbs the bone of the internal nasal crest. He suggested an osteoplastic type of operation which attempted an operation from the frontal sinus to the nose posterior to the duct itself. This did not seem to be effective. Lothrop[19] then described his new external operation which opened both frontal sinuses and left a large opening into the vault of the nose. This was more successful and is still referred to occasionally. Pattee[20] endorsed the Lothrop procedure, although he felt that the external operation was needed only occasionally. Moore[21] believed that more external frontal operations should be done and preferred Lothrop's operation.

Tilley[22] reported a case of acute osteomyelitis of the frontal bone with operation and recovery. It is mentioned specifically because it is apparently the first case of this sort in which simple external drainage was performed early, and then three weeks later, after partial subsidence of the acute phase, extensive bone removal with obliteration of the frontal sinuses was done. This case was not of the extreme fulminating type. It is interesting to consider this report as a precursor of J. M. Brown's teaching somewhat later.

In 1921, Lynch[23] described his operation, stressing removal of the floor of the frontal sinus, complete ethmoid exenteration and complete removal of the sinus lining. This operation at once gained widespread popularity, and all frontal operations through the floor of the sinus have come to be known as Lynch or Lynch-type operations, even though Knapp and Jansen long antedated Lynch.

Harris,[24] discussing reasons for failure of the Killian operation, mentioned three main difficulties: *1.* failure to obtain complete obliteration, especially behind the orbital arch, *2.* reinfection, *3.* contraction of the nasofrontal duct with trapping in the remains of the sinus. In 1923, Skillern,[25] Fielding Lewis[26] and Howarth[27] wrote articles advocating external operation when needed.

Bulson[28] reported a case of apparently spontaneous osteomyelitis which was cured. The patient refused a radical operation at first, so simple external drainage was done. Two and one-half months later a radical operation was done to remove the diseased bone, long after the acute process subsided. This was similar to Tilley's case.

Sewall,[29] in 1926, first reported performing external operations on the ethmosphenoid-frontal group of sinuses under local anesthesia, the chief advantage being a marked decrease in the amount of bleeding. Hurd[30] reported three unusual cases of frontal sinus disease which demonstrated some of the complications and variations that may occur. The first case required multiple operations several years after having a Killian operation because of the development of several mucoceles. The patient finally recovered after the final operation, which was a modified Lothrop procedure to provide drainage into the nose. The second case developed an ossifying periostitis. This case recovered after an obliterative frontal operation. Infected pockets were found trapped within the ossifying tissue. It has some similarity to one of the cases to be reported in this thesis. The third was a traumatic case in which a Killian-Lothrop type procedure was done. Twenty-five days later the patient needed reoperation. The bony bridge of the orbital ridge, the cribriform plate and the posterior wall of the frontal sinuses were all found to be necrotic. The patient then made a slow but uneventful recovery.

Lillie and Anderson,[31] in 1927, advocated a two-stage operation for the frontal sinuses. They advised preliminary intranasal surgery under local anesthesia as the first stage. In 19 of their 60 cases this was sufficient. In the others they proceeded with the second, or external, stage. They obliterated at least the upper portion of the frontal sinus if the front wall was involved, *e.g.*, with a fistula. They felt that this could be readily done by attention to details such as beveling the edges of the bone; furthermore, this, plus avoidance of a vertical incision, helped to lessen the deformity in their cases. In the same year Gill[32] also advocated obliteration rather than an attempt to restore a badly infected sinus.

Mithoefer[33] was one of the first to advocate grafts to line the nasofrontal duct after a Lynch type operation. He used mucous membrane grafts from the nose and even the lip in some cases. Lothrop,[34] in 1928, stated that there were certain objections to all types of radical operations on the frontal sinus. He discussed the difficulty of the occurrence of granulations postoperatively; also, the importance of waiting for a quiet stage in which to perform the radical operation.

A good presentation of gross and microscopic pathology in frontal sinus disease was given by Simpson and Harris,[35] in 1928. They found that there were frequently chronic changes in apparently acute cases, and they particularly noted frequent bony involvement such as osteitis, focal necrosis, osteomyelitis and periostitis.

H. W. Lyman[36] reported an unusual case in 1928. His patient had a chronic left frontal sinusitis and was found to have huge frontal sinuses. A Lynch operation was done on the left frontal sinus which required the use of uterine curettes for removal of the lining. The patient was symptom-free for 20 years before a recurrence. His nasofrontal duct was found to be closed on examination, in 1948; an Ingals gold tube was then inserted to be left in permanent position. This use of the Ingals tube for permanent insertion will be discussed in further detail later.

Lewis,[37] in 1929, strongly condemned all radical sinus surgery as destroying all hope of return to anywhere near normal function. In discussing surgical principles in the treatment of chronic sinus disease, Sewall,[38] in regard to the frontal sinus, cited the desirability but the difficulty of obtaining complete obliteration. As an alternative an attempt is made to get drainage. Here one is beset by the difficulties of closure by scar tissue.

Cullom,[39] after reviewing the history of frontal sinus surgery, concluded that Riedel's operation was too deforming and that Killian's operation was ineffective in obliterating the sinus. He advised taking one's time, doing intranasal work first and then proceeding with a Lynch type of operation.

Lillie,[40] in 1931, presented a discussion of postoperative complications following radical external frontal operations. In a series of 158 cases of either Killian or Lynch operations there were complications in 45 cases with three deaths (two from meningitis and one from brain abscess). Among the nonfatal complications were localized osteomyelitis requiring removal of sequestrums, orbital cellulitis, orbital abscess and erysipelas. Anderson,[41] in a series of 101 cases of either Killian or Lynch operations, stressed two causes of failure: *1.* in the Killian procedure, incomplete operation, resulting in the development of infected pockets; *2.* in the Lynch procedure, contractures of the nasofrontal duct.

In 1933, Mosher with Judd[42] presented his views on treating acute fulminating osteomyelitis of the frontal bone by early radical complete removal of the infected bone with a normal margin, including complete removal of the frontal sinuses.

In the same year Smith[43] described his radical operation in which he exenterated the ethmoids, sphenoids and frontals and then inserted a split thickness skin graft in an attempt to form a new nasofrontal duct. He soon reported[44] a modification of his grafting technique in order to enhance the success of the graft.

Interest in the condition of the lining of the operated frontal sinus was stimulated by the report of Coates and Ersner,[45] in 1930, that in the dog the lining regenerated after its surgical removal; however, as a result of some rather extensive experimental surgery on dogs, Hilding[46] arrived at quite different conclusions. He found that when strips of epithelium were removed they were replaced with heavy scar, which in certain locations interfered with mucus flow, and if near the osteum could block it. If the lining were completely removed, he found[47] that the sinus may obliterate but frequently did not. Some were found to be fairly well restored, but in most of his animals there were scattered cysts filled with mucin in the frontal sinuses. This corroborates the clinical finding of infected pockets found in reoperated cases, a condition which has been reported so many times.

Kleinfeld,[48] in 1934, reported a case of successful collapsing of the frontal sinuses with a Riedel operation. This case had previously had a Killian operation. The obliteration was done because of signs of impending intracranial complications. At the final operation the orbital bridge of bone was found to be partly necrotic. The patient recovered.

Sewall[49] proposed a new type of mucosal flap drawn up into the region of the nasofrontal duct from the lateral nasal wall in an attempt to help form a new duct.

Skillern[50] reported several cases of obliterative frontal sinusitis with infection trapped in the newly growing thick bone. He called this process osteanagenesis. His cases required radical operation with complete removal of the front wall of the frontal sinus.

Diggle and Cawthorne,[51] in 1936, apparently expressing the general trend of thought in England, advocated very conservative treatment of both acute and chronic frontal sinusitis.

In 1936, Reaves[52] demonstrated a practical application of the idea of avoidance of trauma to the nasofrontal duct. In chronic cases, with a fistula, he advised a Killian type of operation leaving a bony bridge, cleaning out the ethmoids into the nose but saving the nasofrontal duct (identified by a probe at operation). He drained his cases externally at first; then the drainage went through the hole in the ethmoids until this scarred up. He felt that by that time the natural frontal duct had recovered its function and could drain the remaining small cavity. This would seem to be a definite advance but was not studied further until some years later.

Adson and Hempstead,[53] in 1937, described an unusual operation for obliteration of the frontal sinus in the presence of osteomyelitis. They used an inverted U-incision, removed the bone from above downward, taking away the posterior wall of the sinus but leaving the anterior wall intact. They tried to remove the mucosa with tincture of iodine. Then the dura and brain came forward into contact with the anterior bony wall to obliterate the sinus. There are several very obvious objections to this procedure, of course. When there is infection in the bone, the anterior wall almost always is involved first. The technical difficulty of removing the sinus membrane would be very great since one of the essential conditions for successful operation is good exposure; furthermore, such wide exposure of the dura left in contact with infected tissue would be highly undesirable. Comments on this procedure have not been offered by any other authors.

In 1937, Mosher[54] said that the frontal sinus operation was still unsatisfactory and that the technique of epidermatizing the enlarged duct or in obliterating the sinus must be perfected. He felt that it would be necessary to remove both anterior and posterior walls to obliterate the frontal sinus successfully. Skillern[55] felt that in cases with osteomyelitic involvement the entire anterior wall of the frontal sinus should be removed.

In 1939, several rather startling ideas were proposed in frontal sinus surgery. None has been generally accepted nor is in use today; however, they are of interest as attempts to solve some of the problems involved. Williams and Fricke[56] proposed the use of radium to keep the nasofrontal opening patent postoperatively but admitted that it was not entirely successful. Matis[57] described a "subperiosteal" operation on the frontal sinus done through the nose and outside the bony edge of the nasal vault. The disadvantage of operating by touch on the frontal sinus seems rather obvious. Kepes[58] proposed a modification of the Killian operation in which he removed the medial half of the bony bridge which Killian originally preserved. Kepes felt that this helped to obliterate the medial portion of the sinus; however, this would make the remaining bone even more prone to necrosis, and photographs accompanying the article show a very marked vertical depression medially. Lothrop[59] insisted that rasping the nasofrontal duct was advisable providing a bony ring was left; however, he then stated that "re-reaming" was easily done subsequently.

Kettel[60] has used a coronal incision from ear to ear, reflecting the scalp downward to expose the frontal bone. Patterson[61] first mentioned the use of chemotherapy in 1939. He felt that prontosil (the first sulfonamide available) made it easier to wait for the best time to operate. He felt that occasionally the anterior wall needed removal, in which case he was careful to bevel the edges of the bone. Jones[62] advocated radical bone removal in cases with osteomyelitis involvement. It is interesting to note that he used the hairline or eyebrow incision and had abandoned the vertical T-incision. Dill[63] advocated more conservative handling of cases of osteomyelitis with early drainage and later removal of sequestrums and obliteration of sinuses if necessary.

Anthony,[61] in 1940, stated that one of the greatest causes for failure in operation, with recurrence of infection, was closure of the nasofrontal opening. He proposed the permanent insertion of a gold Ingals tube to keep the nasofrontal channel open. He reported 10 cases in which this was successful, although, theoretically, this would not be so desirable as a normal duct lined with ciliated epithelium. This idea certainly seems to be a great advance in a certain type of case in which the sinus is not obliterated and yet the duct cannot be saved. This was done in H. W. Lyman's case upon reoperation, as noted above.

Goodale,[65] in 1942, reviewed 182 cases of frontal sinus disease at the Massachusetts Eye and Ear Infirmary in an effort to determine the most important causes of failure in frontal sinus surgery. In 123 cases of infection there were 38 recurrences, or 30.8 per cent. In 18 cases of mucoceles there were four recurrences, or 22.2 per cent. In the cases of infection, four Killian operations were done with three recurrences; 106 Lynch type operations were done with 30 recurrences; and 18 obliterative operations were done with five recurrences. In the cases of mucocele 11 Lynch type operations were done with three recurrences; and seven obliterative operations were done with no recurrences. He found that the three most important conditions causing recurrence necessitating secondary operation were: *1.* scar tissue, *2.* remnants of frontal sinus floor, *3.* ethmoid extension; therefore, he advised that in either obliterative or drainage operations consideration must be given to complete removal of the frontal sinus floor and the ethmoid cells. In 1942, Fred[66] reviewed 40 cases of fulminating osteomyelitis at the same hospital. He found that lower mortality came with early complete removal of the frontal bone; however, he predicted that the sulfonamides would perhaps convert fulminating cases of osteomyelitis into slowly progressive or localizing types, thus permitting more conservative treatment, *i.e.*, the use of sulfonamides with early drainage through the floor and later removal of sequestrums if necessary. Wails[67] advised conservative handling of osteomyelitis following frontal sinusitis. He used early drainage of the sinus followed by removal of sequestrums. He later operated on the sinuses, usually using a Lynch type procedure, occasionally a Riedel type. Jones[68] warned that sulfonamides might mask the clinical course of osteomyelitis and reported a case that died suddenly of a brain abscess. He felt that osteomyelitis of the frontal bone should, therefore, be handled radically as he had previously advocated.

Reporting on a series of surgical experiments on the frontal sinus in dogs, Walsh,[69] in 1943, made some very important observations. He had three groups of animals. In Group I, the nasofrontal duct was enlarged and the rest of the sinus was left untouched. In Group II, the entire sinus was cleaned out and the duct was enlarged. In Group III, the sinus was cleaned out but the nasofrontal duct was left untouched. After the animals were sacrificed, he found that in Groups I and II the sinuses were grossly and microscopically infected. In Group III the sinuses were clean, with completely regenerated lining present. He suggested that there might be a similar response in man as a reason for failures in frontal sinus surgery. He reported three cases of chronic frontal sinus infection in which obliteration was not deemed indicated and in which a Lynch type operation, modified by leaving the nasofrontal duct alone, was done with apparently good results. This is a logical extension of Hilding's work and seems to be an important advance in frontal sinus surgery. Four of the cases discussed in this paper were successfully treated in this manner. The classification of surgical cases into three main categories, of which Walsh's type is one category, will be discussed later.

In 1944, there were four articles published on the use of penicillin in the treatment of osteomyelitis resulting from frontal sinusitis. Iglauer,[70] Kirby and Hepp,[71] Putney[72] and Colbert[73] all agreed that penicillin combined with adequate surgical treatment offered great hopes of reducing the mortality of the disease and permitting more conservative surgical management. They also found that the sulfonamides were relatively ineffective. Schnitker and McCarthy[74] confirmed these findings in 1945, but emphasized that penicillin is not a substitute for surgery. They found that surgical excision of diseased bone was necessary in most instances and that it could be done as soon as the infection was controlled by penicillin, at which time primary closure of the scalp was a feasible procedure.. They then performed a secondary operation for correction of the bony defect three months later, using a tantalum plate.

Discussing causes of failure in the surgical treatment of chronic sinusitis, Russell[75] stated that long standing cases with high grade degeneration of the mucous membrane, denuded bone, necrotic areas and fistulous tracts were unsuitable for drainage and needed complete obliteration of the frontal sinuses. He said that by far the most satisfactory of the obliterative operations was Riedel's, and that although the deformity was severe, corrective plastic surgery was improving. He felt that only a small percentage of cases needed obliteration, but in those that did, one should not temporize. Goodale,[76] in 1945, suggested the permanent placement of a sheet of tantalum foil for the formation of a nasofrontal duct after the Lynch operation. This idea has received considerable favorable attention. Jesberg[77] felt that complete obliteration of the frontal sinus produced too much deformity and proposed an operation in which the lateral half of the sinus was obliterated, converting it into a small cavity which he felt was easier to drain. This is essentially a compromise. Van Alyea[78] strongly advised conservative surgical measures directed toward institution of adequate sinus drainage. He felt that radical removal of sinus mucosa was seldom indicated.

Boise,[79] in 1942, strongly advocated early trephining through the floor of the sinus in acute frontal sinusitis as a measure to prevent the development of complications such as osteomyelitis and intracranial extension. This idea was emphatically seconded by Brown,[80] who also said that penicillin was very useful but did not remove pus already present. After the acute process has subsided, Brown advised a Lynch type external operation leaving the nasofrontal duct intact according to Walsh's idea. Goodyear[81] also favored early trephining in acute frontal sinusitis. He further recommended the permanent insertion of Ingals' gold tube in all external frontal operations, just as had Anthony a few years before. In attempting to obliterate certain frontal sinuses, Goodyear modified the Killian operation by packing the skin down into what was left of the frontal space. One would expect marked deformity with such a maneuver.

In 1946, Weille[82] reported a new series of cases at the Massachusetts Eye and Ear Infirmary. There were 276 cases, requiring 862 operations. In the Lynch operations closure of the nasofrontal duct was the overwhelming cause of failure. He was impressed by Goodale's tantalum foil and Walsh, Brown and Hilding's idea of leaving the duct alone as promising attempts to remedy this cause of failure. He found that in the obliterative operations, besides the drawback of deformity, frequently the sinus had not actually been obliterated. Mosher[83] summaried his views on frontal sinus surgery in "My Milestones." He reiterated his advice to respect the virginity of the nasofrontal duct because if traumatized it always scars down. He mentioned the work of Hilding and Walsh. He felt that in acute frontal sinusitis early external trephining as advocated by Brown was the best procedure. He discussed the difficulties of the various frontal sinus operations and said he had never been satisfied with any of them. He felt that if the frontal sinus required obliteration, it would be best to remove both anterior and posterior walls. Finally he felt that antibiotics were a tremendous aid in treating osteomyelitis, but that if edema was present and did not regress in 48 hours extensive bone removal should still be practiced.

Negus[84] presented his views on frontal sinus surgery in 1947. In the external operation he used a small incision, did not remove all of the floor, did not curette out the membrane and enlarged the nasofrontal duct, using a skin graft. This is in direct contrast to the opinions found in current American literature. In the same year Erich and New[85] presented an acrylic obturator which they inserted into the nasofrontal passage a few days postoperatively and left in place for six months. They felt that this gave the passage time to become epithelialized. Besides the obvious objection to wearing a bulky obturator which blocks one side of the nose for six months, there remains the question whether this new passage would not eventually contract and close. Bergara[46] described a very complicated "osteoplastic" operation, making a flap of the anterior frontal sinus wall which he replaced at the completion of the operation. He had nothing to contribute to the problem of dealing with the nasofrontal duct. This type of operation has been reported several times but has never gained acceptance in this country.

In recent years there have been a good many reports of various methods of repair of operative defects in the frontal bone. O'Connor[47] recommended the use of preserved isocartilage grafts. New and Dix[48] used autocartilage grafts from the thoracic cage. Kazanjian and his associates[49,50] have used bone, cartilage, vitallium, tantalum and acrylic. They found that each material had certain advantages and disadvantages, hence they individualized their cases. Even more recently Peer[51] has used diced cartilage grafts and Lierle and Huffman[52] have used autogenous cancellous bone broken up into minute bits. These last two methods are claimed to have the advantage of ease of molding into the desired shape and avoidance of subsequent ridges or bumps.

In January, 1949, Arbuckle[53] reported seven cases of chronic frontal sinusitis with complications, of which six required complete obliteration of one or both frontal sinuses. Winborn[54] advised the following measures in the management of severe frontal sinus infections: heavy doses of penicillin; early trephining of the floor; and later, after subsidence of the acute process, an external operation, usually some type of modification of the Killian, to attempt to obliterate the sinus.

Since this thesis was orginally submitted, Work[55] published an instructive article about the relation of the frontal sinuses to skull injury. He found that in cases in which tantalum plates had been inserted infection was inevitable if the plate was in contact with the frontal sinus. He also noted that tantalum plates in the forehead were frequently subject to trauma and rather easily became bent. He strongly advised complete obliteration of the frontal sinuses prior to insertion of a tantalum plate for cosmetic repair of frontal defects.

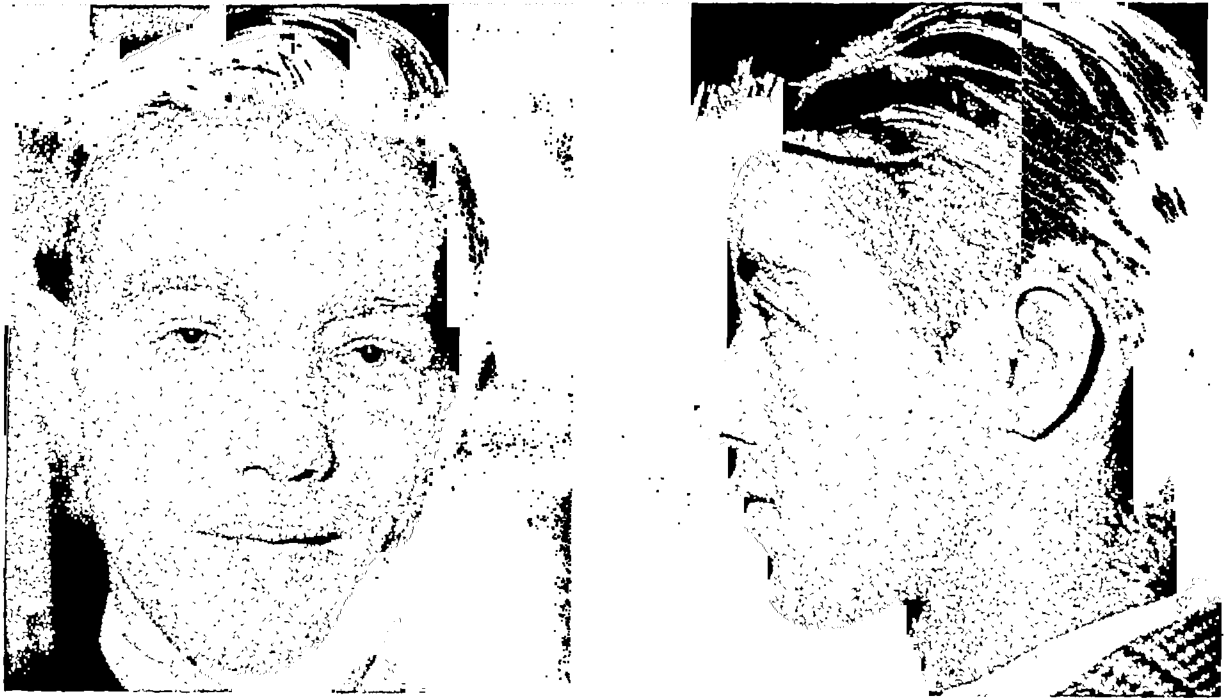
III. SUMMARY OF HISTORICAL REVIEW.

Since the literature is voluminous it is advisable to give a summation. Ogston first opened the frontal sinus through the anterior wall for drainage in 1884. Riedel described complete operative obliteration of the frontal sinus in 1898. Apparently because of the deformity and the risk of the operation it did not become widely used. Killian, in 1903, described his operation and this was immediately greeted with a wave of enthusiasm. The enthusiasm soon abated as the high operative mortality and the frequency of recurrence became apparent. Knapp first entered the frontal sinus through the floor in 1908 and this procedure was popularized, in 1921, by Lynch. It gradually was realized that this operation, too, was prone to lead to recurrence, chiefly because of closure of the nasofrontal opening. It was further gradually realized that radical frontal sinus surgery should not be done in the acute phase of infection except in the presence of a fulminating osteomyelitis. Many attempts have been made to solve the problem of postoperative closure of the nasofrontal duct. Ingals introduced his gold tube for temporary insertion in 1906. It was not until 1940 that Anthony proposed permanent placement of the Ingals tube in the nasofrontal duct region. Goodyear also advised this in 1947. In 1942, Goodale suggested the permanent insertion of a piece of tantalum foil. In 1933, Hilding reported his own experiments showing scarring and closure of the nasofrontal duct. In 1936, Reeves first proposed leaving the nasofrontal duct alone in external frontal sinus surgery. This was followed by Walsh who presented experimental and clinical evidence to further this idea in 1943. Boies, in 1942, and shortly afterward Brown and Goodyear, all strongly advised early trephining of the floor of the frontal sinus in acute infection. This idea has become generally accepted. Many other ideas of variations in operative technique and procedure have been proposed and discarded. In the last 10 years there have been a good many articles dealing with plastic repair of defects of the frontal bone. In the last few years reports have been presented discussing the use of penicillin in frontal sinus disease and its complications. Penicillin has been found to alter the prognosis of serious cases favorably and allow a more cautious and studied approach to the proper surgical management of difficult cases. It has been found to reduce the incidence of radical operation but the antibiotics are not a substitute for surgery. It has enabled, generally, more conservative management of osteomyelitis. Although most authors seem to have tried to avoid the Riedel obliterative operation, there have been occasional reports through the years of its successful use.

IV. CASE REPORTS.

The following three cases are presented with pertinent comments in each instance. Pictures, X-rays and microphotographs are illustrated. One case is reported from each of the three types of cases. Thirteen of the cases were patients at the Veterans Administration Hospital, Jefferson Barracks, Mo.; four cases are from other hospitals.

Case 1: F. V., a 21-year-old white male entered the Veterans Administration Hospital on the evening of Sept. 5, 1947, on the emergency surgical service and was transferred to otolaryngology the next morning. He gave the history of having had a cold a week previously. Four days before admission he noticed a boil on the back of his neck and severe right frontal headache which prevented sleep. For the past two days he had noticed increasing swelling of the right eye. He also noticed aching in the right upper molar teeth. He had photophobia and nausea. In his past history he had been subject to boils but had never had any headaches. Examination showed a young asthenic man severely and acutely ill. His temperature, on admission, was 101.8° F. There was diffuse edema extending about half way up the forehead. The right upper eyelid was red and very swollen. There was chemosis of the right eye. The nasal mucosa was acutely inflamed and very swollen. There was thick yellow pus in both nasal fossae. There was a sharp deviation of the septum to the right. There was a draining furuncle on the back of the neck. He was started on 50,000 units of penicillin every three hours on admission. The next morning this was increased to 100,000 units every three hours, and he was then started on 1 gm. of sulfadiazine every four hours with an initial dose of 4 gm. The edema of the forehead continued to increase and reached the hairline during the morning after admission. It was felt that he had an acute fulminating right frontal sinusitis with spreading osteomyelitis; therefore, the floor of the right frontal sinus was trephined under local anesthesia at noon on Sept. 6, 1947. The opening



Figs. 1 and 2. Case 1. Front and lateral views showing postoperative appearance following obliteration of the frontal sinuses.

was enlarged with mallet and gouge to a diameter of one-half cm. A small amount of liquid pus was obtained. A flat rubber drain was inserted and a dressing applied. The patient's condition remained stationary for about 24 hours and then began to improve rapidly. The pain, swelling, fever and headache rapidly subsided.

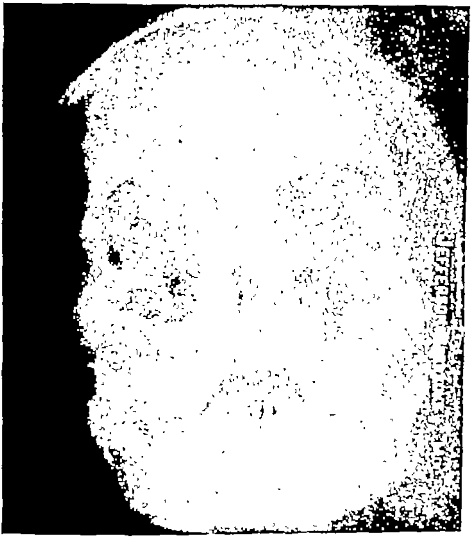


Fig. 1. Case 1. Preoperative X-ray in Waters' position with lipiodol showing extreme thickening of mucosa in frontal sinuses and blocked right nasofrontal duct.

On Sept. 25, 1947, a submucous resection was done to improve the airway and facilitate intranasal drainage. On Oct. 1, 1947, the rubber drain in the right frontal sinus was removed. After the patient's condition had improved, extensive X-ray studies, *including lipiodol films*, were done to help determine the condition of his sinuses and frontal bone. X-rays showed a pansinusitis on the right side and clouding of the left frontal sinus. The right antrum was clear on irrigation and finally cleared on X-ray. Lipiodol could not be instilled into the right frontal even through a cannula in the nasofrontal duct. Oil instilled in the left frontal sinus showed very thickened irregular lining. The X-rays did not reveal the true extent of bone damage as found at operation. The sulfadiazine was discontinued on Sept. 23, 1947.

On Nov. 6, 1947, a bilateral radical obliterative frontal operation was done under intravenous sodium pentothal anesthesia supplemented with local infiltration with 1 per cent novocaine. A horizontal incision just below the eyebrows was made and the periosteum elevated. A dehiscence, about 1 cm. in diameter, was found in the anterior wall of the left frontal sinus. On the right side the surgical opening through the floor was uncovered. The anterior wall of both frontals was rough, soft and spongy and was removed completely. The membrane was quite roughened and thickened. This was completely removed. The floor of both frontals was removed and the anterior ethmoids were cleaned out. There was thickened membrane in the ethmoids, but frank pus was found in only two of the left anterior ethmoid cells. The bone at the root of the nose and laterally about the upper outer rim of the orbits was taken down. Rubber drains were placed laterally on each side and brought out at the root of the nose. The periosteum was closed with chromic catgut, the subcutaneous tissue with plain catgut, and the skin with silk. A pressure bandage was applied. Specimens of tissue removed at operation revealed chronic osteomyelitis and chronic sinusitis. His postoperative

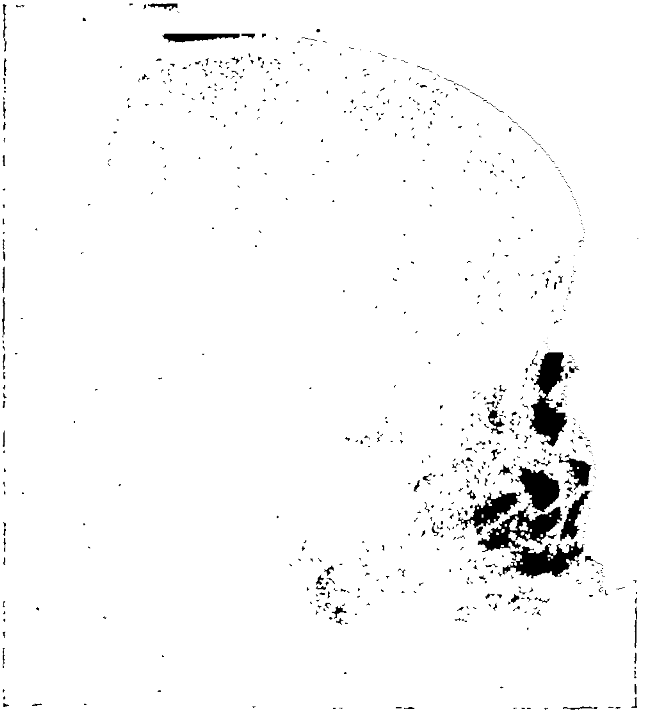


Fig. 4. Case 1. Postoperative X-ray in lateral view showing obliteration of the frontal sinuses and the extent of bone removal.

course was completely uneventful. The drains were removed on the third postoperative day and the sutures on the fourth and sixth postoperative days. He developed a transient diplopia which rapidly disappeared. Penicillin was stopped on Nov. 17, 1947. The patient left the hospital on leave of absence on Nov. 19, 1947, and returned for periodic checkups until he was given his final discharge on Dec. 20, 1947. He has been completely well since that time.

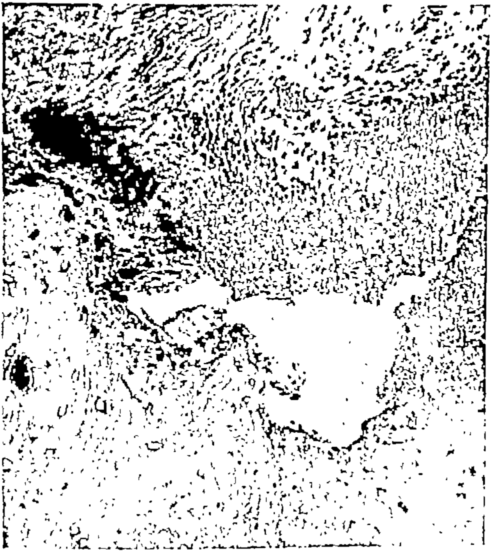


Fig. 5. Case 1. Microphotograph of piece of bone from anterior wall of frontal sinuses showing chronic osteomyelitis.

Comment: This case illustrates the acute fulminating type of infection with early development of osteomyelitis as shown by the spreading edema of the forehead and the extreme toxicity of the patient. Even with heavy doses of penicillin the patient became worse in a matter of hours until an external trephine was done through the floor of the right frontal sinus, On Nov. 6, 1947, a bilateral radical obliterative frontal operation was done under intravenous sodium pentothal anesthesia supplemented with local infiltration with 1 per cent novocaine. A horizontal incision just below the eyebrows was made and the periosteum elevated. A dehiscence, about 1 cm. in diameter, was found in the anterior wall of the left frontal sinus. On the right side the surgical opening through the floor was uncovered. The anterior wall of both frontals was rough, soft and spongy and was removed completely. The membrane was quite roughened and thickened. This was completely removed. The floor of both frontals was removed and the anterior ethmoids were cleaned out. There was thickened membrane in the ethmoids, but frank pus was found in only two of the left anterior ethmoid cells. The bone at the root of the nose and laterally about the upper outer rim of the orbits was taken down. Rubber drains were placed laterally on each side and brought out at the root of the nose. The periosteum was closed with chromic catgut, the subcutaneous tissue with plain catgut, and the skin with silk. A pressure bandage was applied. Specimens of tissue removed at operation revealed chronic osteomyelitis and chronic sinusitis. His postoperative

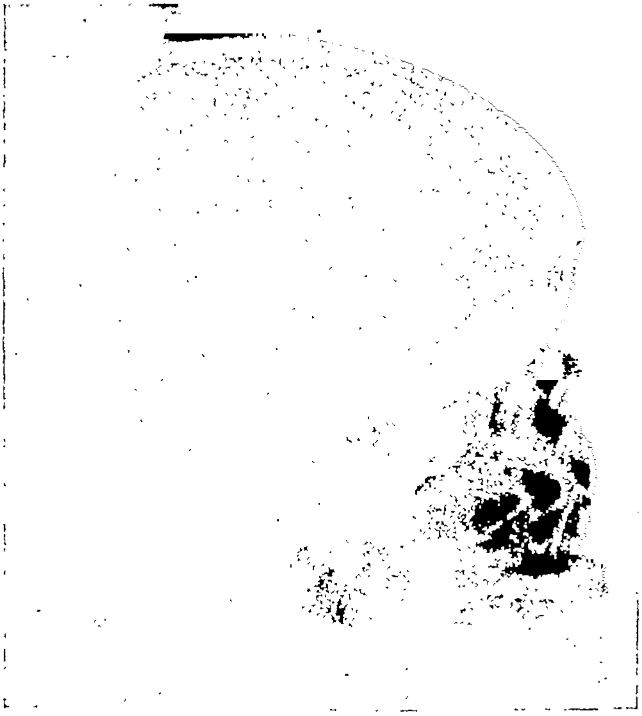


Fig. 4. Case 1. Postoperative X-ray in lateral view showing obliteration of the frontal sinuses and the extent of bone removal.

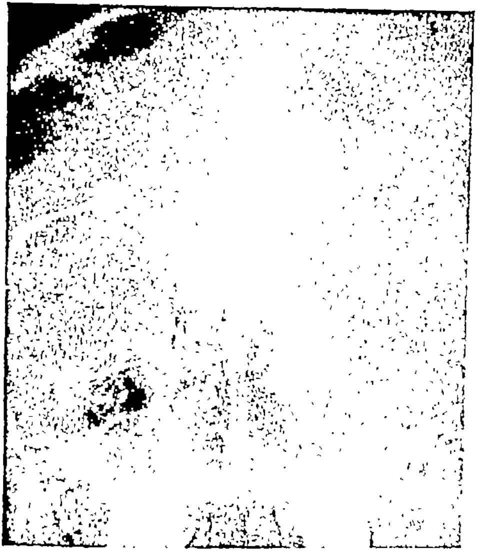


Fig. 6. Case 2. X-ray in front view showing the position of the tube. The extremely large size of the frontal sinuses can be seen, as can the thickening of the sinus membrane.

in the bony walls to warrant obliteration had to be considered; however, the extremely large size of the frontals, combined with the fact that this paient had already had several plastic operations for the repair of the left maxilla, which had been shattered by shrapnel, and the fact that he was symptom-free as long as the nasofrontal duct remained open led to the use of the procedure of permanent insertion of the gold Ingals tube.

Case 3: H. W., a 40-year-old white male, entered the Veterans Administration Hospital on March 25, 1949, with the complaint of right frontal headaches for five years. In 1944, during a search for foci of infection because of backache, an infection of the right maxillary sinus was found and an antrum window was made. He got along fairly well until about one year before admission when the headaches began to recur. They gradually became worse and he finally came to the hospital. Examination revealed pus in the right middle meatus, streaming over the right inferior turbinate. There was polypoid tissue hanging over the right middle turbinate. The previous antrum window was found to be closed.

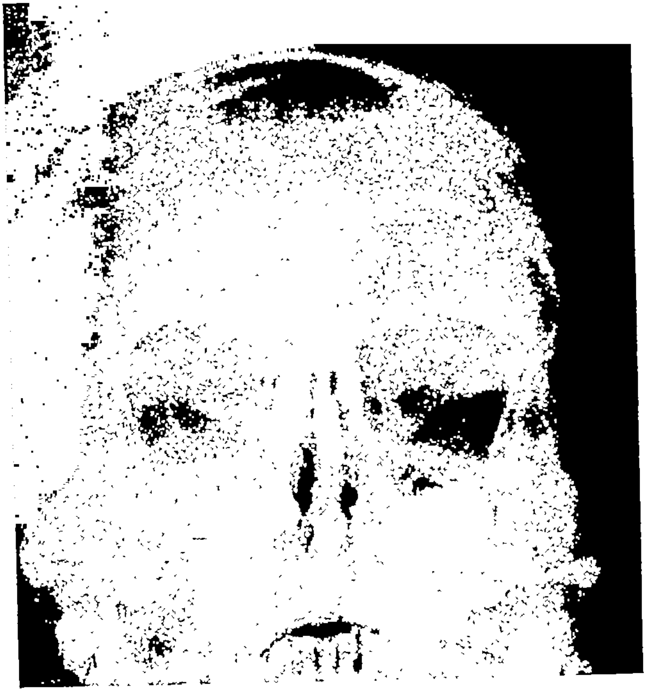


Fig. 7. Case 3. X-ray in Waters' position showing clouding of right frontal sinus with sharply defined area of sclerosis adjacent. Lipiodol in antrums shows thickened lining in the right antrum.

Pus was obtained on antral irrigation. Culture yielded hemophilus influenzae. Sinus X-rays revealed a pansinusitis on the right side with a well defined area of heavy sclerosis about the right frontal sinus. Lipiodol films showed marked thickening of the lining of the right antrum. There was thickened membrane in the right frontal sinus, but the nasofrontal duct was patent.

A right Caldwell-Luc operation, with transantral ethmoidectomy and polypectomy was done on April 4, 1949. The antrum was full of pus and lined with badly infected thickened lining. The lining of the ethmoid cells was thickened with polypoid degeneration. The patient was given 50,000 units of penicillin every three hours from April 5, 1949, to April 8, 1949. His postoperative course was uneventful. Penicillin was resumed on April 20, 1949, and stopped on April 27, 1949. On April 20, 1949, a right modified Lynch type operation was done under local anesthesia. A gently curved incision was made through the shaved brow on the right side to and through the periosteum. The right frontal sinus was entered through the floor. The bone was smooth, hard and thickened. Pus was found in the sinus and the mucosa was irregular and thickened. There was a sharp line of demarcation in the mucosa near the entrance to the nasofrontal duct which was patent. The mucosa, at the entrance of the duct, was normal. The mucosa, at the line of demarcation, was divided with a sharp knife and the lining of the sinus completely exenterated, leaving the nasofrontal duct and its entrance intact. There was a small lateral extension of the frontal sinus filled with infected granulations. This was completely exenterated. The floor of the sinus was completely removed. A small rubber drain was placed in the sinus and brought out through the medial end of the incision. The soft tissues were closed with catgut and the skin with silk. A small dressing was applied. The drain was removed on the third postoperative day and the sutures on the fifth. The patient's postoperative course was completely uneventful, and he left the hospital on April 29, 1949, completely relieved of his headaches. He has remain symptom-free since that time.

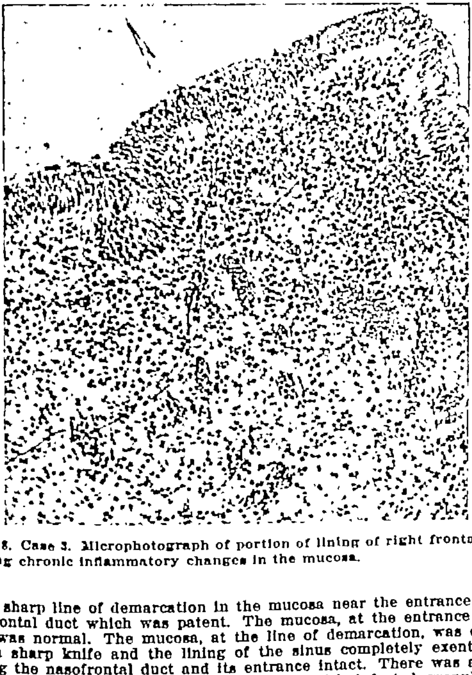


Fig. 8. Case 3. Microphotograph of portion of lining of right frontal sinus showing chronic inflammatory changes in the mucosa.

Comment: This case illustrates the type of sclerosing sinusitis in which the bone surrounding the sinus becomes hypertrophied and is apparently attempting to obliterate the sinus. Certainly there was no evidence of infection in the bone itself and the process may be considered a protective mechanism; however, it is noteworthy that there was a tract filled with infected tissue projecting laterally from the main portion of the right frontal sinus. The sharp demarcation between obviously infected sinus membrane and relatively normal mucosa leading into the nasofrontal duct was also very striking. This was felt to be an ideal case for a Lynch type operation as modified by Walsh and others to leave the nasofrontal duct intact.

V. DISCUSSION.

Consideration of this series of cases shows that cases of frontal sinus infection requiring surgery lend themselves to classification into three categories. The 11 cases in this series in the category requiring obliteration of the frontal sinuses can be classified into three main groups. Group 1 consists of the traumatic cases, in which infection of the frontal bone and sinuses follows external injury. There were two cases of this type. Group 2 includes the acute fulminating frontal sinus infections in which there is rapidly progressive involvement of the bone. There were two cases of this type. Group 3 includes the cases of chronic infection in which there are repeated acute exacerbations and sooner or later the development of infection in the bone as manifested by draining fistulas, necrotic areas in the bone and marked degeneration of the mucous membrane. There were seven cases of this type.

In all of these cases there was severe infection in the frontal sinus itself with irreversible changes in the mucous membrane plus infection in the bony walls of the sinus. Penicillin and other antibiotics, such as streptomycin, are effective aids in treating these cases but do not take the place of surgery. The antibiotics cannot restore a necrotic membrane to normal and are admittedly not as effective in treating infection involving bone as that involving soft tissue alone. In all the cases in which the frontal sinuses were obliterated, the bone of the front wall was soft and rough, and frequently discolored and necrotic. In contrast, in most of the cases the posterior wall was in good condition. In three of the cases there was a dehiscence in the posterior wall but the rest of the bone of the posterior wall was sound. In one of the traumatic cases almost all of the posterior wall had been destroyed at the time of the accident, or removed at debridement by the neurosurgeon.

In consideration of the proper time for operation, in the acute cases early drainage of the frontal sinuses through the floor is very important. Then a period of antibiotic therapy and observation for subsidence of the acute infection is essential. This should take anywhere from three to six weeks, or longer, depending upon the individual case. If the pathological process is well localized, or is chronic when first seen, the waiting period may not need to be as long.

Clinically, all of the patients whose frontal sinuses were obliterated have done well, with no evidence of reinfection in the frontal region. One case required a long period of observation for possible neurological disease but finally became completely symptom-free. None has shown any evidence that the frontals were not completely obliterated.

Another interesting fact is the presence of severe infection bilaterally in almost every case. In only two cases was one of the sinuses much less involved than the other. In these cases the mucosa was only moderately thickened and the nasofrontal duct was recognizable as such on the left side in contrast to the necrotic tissue in the right side with softening of the anterior wall.

Cosmetically the best results have been obtained by using a horizontal incision gently curved to go through the brow with the connecting portion of the incision over the bridge of the nose at or near the frontonasal suture. The incision is curved downward from the inner end of the brow and then turned at a right angle to go over the bridge of the nose. Special attention is given to beveling the edges of the bone, trimming down the upper and outer edges of the rim of the orbit, removal of any supraorbital extension of the frontal sinuses posteriorly and exenterating all or part of the ethmoid labyrinths as needed. Gelfoam or oxycel is placed in any dead space which may be found remaining in the central portion of the operative field. Primary closure of the incision is practiced, with long, narrow, flat rubber drains placed to come out at each end of the incision. These are gradually shortened and are removed in a few days. Great care must be taken to approximate the skin edges accurately and smoothly about the bridge of the nose. The periosteum is sutured with interrupted chromic catgut and the skin with interrupted silk sutures. A moderate pressure bandage is kept on the forehead for about one week, being changed as needed. Mechanic's waste is a good material to include in the bandage since it exerts gentle pressure. The skin sutures are removed in four to six days. There is considerable postoperative edema of the upper eyelids but this subsides without difficulty. Transient diplopia has been fairly frequent immediately postoperatively but has not been permanent in any of these cases.

All of the patients were men. In women, the cosmetic aspect requires more attention, but with recent improvements in plastic surgery the main issue of dealing with the infection, which may be fatal if unchecked, should not be sidestepped.

The small amount of constitutional reaction to this major operation is surprising; however, the patient is carefully prepared for surgery. Intravenous fluids, and whole blood if deemed advisable, are used during the operation. Penicillin is used intensively both pre- and postoperatively. Other antibiotics, or one of the sulfonamides, may sometimes be used to supplement the penicillin. Various anesthetics have been used, usually including local infiltration with novocaine and adrenalin plus either sodium pentothal, intravenously, or occasionally nitrous oxide and ether endotracheally.

Admittedly, the cases of frontal sinus infection which need obliteration do not occur very frequently in relation to the total number of cases; however, when the occasion does arise it would seem best to study the patient carefully, prepare him for operation and then proceed to definitive treatment and not try to temporize. These cases have been presented as illustrations of this viewpoint.

The question arises what to do about other cases in which obliteration is not indicated. It would seem that a certain number of cases would come into the second category, in which there is definite infection in the sinuses, causing disabling symptoms, not relieved by conservative treatment but showing no evidence of extension of infection to the frontal bone itself. There were four of these cases in the series. These cases seem to be well handled by the Lynch procedure as modified by Walsh, Hilding and Reeves to leave the nasofrontal duct intact.

This leaves a third category of cases: those in which the nasofrontal duct has been destroyed, either by disease or by previous operation, and in which obliteration is not indicated or feasible. Case 3 illustrates this type. There was one other similar case, in which the duct had been closed off by a shrapnel wound. Here the insertnon of a gold Ingals tube, to be left in place permanently, seems to be the best solution, as suggested by Anthony and Goodyear; Goodale's tantalum foil is probably an alternative that would be just as effective. Although these cases do not have a duct with ciliary action, apparently gravity assisted by occasional blowing of the nose is amply sufficient to provide drainage and prevent blocking of secretions.

VI. CONCLUSIONS.

1. The literature of frontal sinus surgery is voluminous and controversial.

2. Penicillin is a marvelous aid in treating frontal sinus infections, but is not a substitute for necessary surgery.

3. Cases of frontal sinus infection requiring surgery can be classified in three categories.

4. There are certain cases which still occur that need complete obliteration of the frontal sinuses for cure. These are considered as belonging to the first category.

5. These cases all have extension of infection into the frontal bone with irreversible changes and may be classified in three groups: Group 1, traumatic; Group 2, acute fulminating infections with spreading involvement of bone and impending complications; Group 3, chronic infections with low grade osteomyelitis as exemplified by the presence of chronic draining fistulas, small areas of necrotic bone and extreme degeneration of the mucous membrane. The cases in Group 3 frequently are characterized by repeated acute exacerbations.

6. If obliteration of the frontal sinuses is indicated, the complete obliterative operation should be done. This includes complete removal of the anterior wall and floor of the frontal sinuses and all their contents, but need not include the posterior wall unless it shows definite pathological changes or there is evidence of intracranial extension of the infection. If the ethmoid labyrinths are involved they should be exenterated as well.

7. The cosmetic result following the obliterative operation can be good if careful attention is paid to the incision and to beveling down the bony margins and the upper outer rim of the orbit to avoid a dish-face appearance. The inverted T-incision has been abandoned and a gently curving horizontal incision in the brow is used. The incision is closed primarily with careful attention to the proper approximation of tissues, especially about the root of the nose. Long, narrow drains are placed at the outer ends of the wound. A moderate pressure bandage is applied. If the patient feels that he wants a plastic operation later, recent developments in plastic surgery offer several methods of correcting the bony defect.

8. Certain other cases of frontal sinusitis in which the bone is not involved are well treated by the Lynch operation as modified to leave the nasofrontal duct intact. These are considered as belonging to the second category.

9. There are a few cases coming to surgery which are not covered by either of the above categories. These include cases in which the nasofrontal duct has been destroyed by disease or previous operation but in which obliteration is not indicated. These cases are best treated by the permanent insertion of the gold Ingals tube. These are considered as belonging to the third category.

10. The classification of cases of frontal sinus infection requiring surgery into these three categories for consideration of the proper surgical approach appears logical and is based on the pathological conditions present and on the most rational method of correcting the condition in each type of case.

VII. SUMMARY.

The literature of frontal sinus surgery is reviewed. Three case reports are presented. The first illustrates obliteration of the frontal sinus, performed in 11 cases. The second illustrates permanent insertion of the gold Ingals tube, performed in two cases. The third illustrates preservation of the frontal sinus with an intact nasofrontal duct, performed in four cases. Various aspects of the operations are discussed and certain conclusions drawn.

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308 North 6th Street.

THE MANAGEMENT OF TINNITUS.*†

VICTOR GOODHILL, M.D.,
Los Angeles, Calif.

I. INTRODUCTION:

A reorientation is necessary in our attitude toward the management of tinnitus. Tinnitus is a symptom, not a disease. It is, therefore, unscientific to speak of the treatment of a symptom of almost any otologic disease. There can be no proper discussion of the treatment of tinnitus as a pathologic entity. It is unreasonable to expect any one method of treatment, drug or operation, to favorably influence a symptom which may arise from a host of causes in a number of different locations in the auditory pathway.

The patient who suffers from tinnitus, however, deserves much of our thought. The management of the problem presented in a given patient is a sober challenge.

II. TERMINOLOGY AND DEFINITIONS:

The terminology employed in the discussions concerning tinnitus has been very confusing and has resulted in much of the lack of clarity on this subject.

The word tinnitus, derived from the Latin, refers to a jingling or tinkling. It is necessary immediately to distinguish two separate applications of the term tinnitus, namely, tinnitus aurium and tinnitus cerebri (tinnitus cranii). The former term refers to tinnitus of ear origin and the latter to tinnitus of intracranial origin. The two types of tinnitus are

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif., Jan. 21, 1950.

†From the Department of Otolaryngology, University of Southern California School of Medicine, Los Angeles, Calif.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

entirely different in location, but they may co-exist. Accordingly, the first hurdle in the terminology is the distinction between these two basic types of sensory phenomena.

Tinnitus aurium is usually localized to the one ear or both ears, and described by the patient in some specific manner, *i.e.*, steam escaping, seashell, ocean roar, bells ringing, whistles, click, rumble, static, etc.

Tinnitus cerebri is not too well localized by the patient. It is in the head and usually is described as a generalized roaring. It is rarely a specific type of noise. It is diffuse and may even be attributed to a roaring in the neck and occasionally thorax. It is usually unaccompanied but may occasionally be accompanied by tinnitus aurium. It properly belongs in the domain of the general physician or neurologist. It may be due to vascular intracerebral disease and usually has same psychosomatic components. Occasionally it is difficult to distinguish between tinnitus cerebri and tinnitus aurium.

Tinnitus aurium must be divided into two distinct categories, which may be defined as follows:

1. *True or subjective tinnitus* is the subjective cortical perception of auditory sensations inaudible to anyone but the

TABLE A.

TINNITUS	
STATIC	DYNAMIC
NONVIBRATORY	VIBRATORY
TRUE	PSEUDO
SUBJECTIVE	OBJECTIVE
INTRINSIC	EXTRINSIC
COMPENSATED	
UNCOMPENSATED	

patient. Such sensations may be due to an actual subaudible somatic source, or as is most generally the case, to an auditory paresthesia from anywhere in the auditory pathway.

2. *Pseudo or objective tinnitus* is the subjective cortical perception of auditory sensations potentially audible to an examiner, as well as to the patient.

These two subtypes have been given several synonyms, which are summarized in Table A.

III. PSEUDO OR OBJECTIVE TINNITUS:

This type of tinnitus has been characterized by Fowler as "vibratory." Numerous cases have been reported in the literature, but in general, this type of tinnitus is relatively *very uncommon* as compared to the extremely common subjective or true tinnitus. Pseudo or objective tinnitus is usually either vascular or muscular in origin.

The vascular phenomena responsible for pseudo or objective tinnitus are usually connected with organic vascular disease, most frequently characterized in the intracranial aneurysm. This type produces an auditory murmur which is quite audible to an examiner.

Muscular causes for pseudo or objective tinnitus will usually arise from either the tensor tympani, stapedius or salpingopharyngeus muscles. These muscular clicks are easily audible by an examiner. Frequently the audible click may be accompanied by a visible phenomenon in the tympanum or in the tubal area. The mechanism underlying these unusual muscular contractions is not well understood. It has been presumed that psychogenic factors of the "tic mechanism" variety play a great part in the origin of the muscular variety of objective tinnitus. Fatigue factors and involuntary reflex muscular contractions may play a part in the causation.

IV. TRUE SUBJECTIVE TINNITUS — DIAGNOSTIC ASPECTS:

The patient with true subjective tinnitus presents a double diagnostic problem. The first aspect of the problem is the etiologic diagnosis, and the second aspect is the psychosomatic diagnosis. A valid assessment of any patient presenting the symptom of true or subjective tinnitus demands that the diagnosis be dual; namely, etiologic and psychosomatic.

1. *Etiologic Diagnosis:*

The etiologic diagnosis must be based upon *a.* anatomic localization and *b.* pathologic physiology. As illustrated in Table B, the anatomic location may include any portion of the auditory pathway from the external auditory canal to the auditory cortex. The pathologic lesion may be of almost any type of derangement peculiar to the anatomic site. A brief

TABLE B.

ETIOLOGY OF TINNITUS (DIFFERENTIAL DIAGNOSIS).

ANATOMIC LOCATION	PATHOLOGIC LESION
EXT. AUD. CANAL	Anemia
TYMPANUM	Hyperemia
a. Memb. tymp.	Edema
b. Tymp. muscles	Serous exudate
c. Ossicles	Mucus exudate
d. Tymp. plexus	Purulent exudate
e. Tymp. vessels	Hemorrhage
f. Eust. tube	Inflammation
g. Fenestrae—Rotund Ovale	Allergy
COCHLEA	Neuronitis
a. Perilymph	Necrosis
b. Endolymph	Fibrosis
c. Organ of Corti	Tumor
VIIIth NERVE	Aneurysm
a. Spiral ganglia	Capillary fragility
b. Trunk	Stasis
INTRACEREBRAL	Sludging
a. Ventral and dorsal nuclei	Vascular spasm
b. 2nd order neurons	Vascular sclerosis
c. Med. gen. body	Vasomotor paralysis
d. 3rd order neurons	
e. Cortex	

listing of anatomic locations and pathologic lesions as shown in Table B illustrates the great complexity of possibilities to be considered under etiologic diagnosis. In addition to the infinite variety of lesions and locations, there are extensive variations in intensity of pathological disturbances and extent of anatomical involvement.

The most common anatomic location is probably in some portion of the organ of Corti and its neuronal extensions. The most common pathologic lesion is probably vascular.

It must be remembered in connection with the etiologic diagnosis that tinnitus is one of the five cardinal symptoms of ear disease, the other four being otalgia, otorrhea, vertigo and deafness.

2. Psychosomatic Diagnosis:

The psychosomatic diagnosis or perhaps, more logically, the "psychosomatic loading factor" is of as great importance in the proper analysis of a patient with tinnitus as the etiologic diagnosis. The psychosomatic diagnosis must be approached from two standpoints: *a.* the emotional threshold or sensitivity level of the patient, and *b.* specific psychodynamic problems. Dr. E. P. Fowler, Sr., should be greatly commended for drawing our attention to this approach and for emphasizing its great practical importance in the management of the patient with tinnitus (see Table C).

TABLE C.

TINNITUS IS A DOUBLE DIAGNOSTIC PROBLEM.	
ETIOLOGY—DIFFERENTIAL DIAGNOSIS	PSYCHOSOMATIC WEIGHT (LOADING FACTOR)
a. ANATOMIC LOCATION	*a.* EMOTIONAL THRESHOLD (sensitivity level)
b. PATHOLOGIC PHYSIOLOGY	*b.* SPECIFIC PHOBIAS (anxiety states, conversion mechanisms)

A. Emotional Threshold—Sensitivity Level of Patient:

There is no correlation between the actual intensity of tinnitus as measured audiometrically and the amount of suffering of the patient. It is important, therefore, to attempt to assess the excitability level of the patient. This is not a simple matter and requires keen clinical judgment.

B. Specific Psychodynamic Problems:

The specific psychodynamic problems that must be considered are primarily in the nature of phobias, anxiety states and conversion mechanisms. Some of the most common problems are those involving the fear of impending insanity, the fear of a brain tumor, and the fear of total deafness, particularly if the patient already has a significant hearing loss.

V. TINNITUS ANALYSIS—RECOMMENDED PROCEDURE:

1. History:

A chronological detailed history is of prime importance and need not be stressed further.

2. Otologic Examination:

The otologic examination should include, in addition to a thorough physical examination of the ears, nose and throat, pure tone and speech audiograms, recruitment tests, tinnitus localization on the audiometer, both as to intensity and frequency, and caloric vestibular tests.

3. Simple Medical Findings including blood pressure, fundus examination, hemoglobin determination and a rough neurological screening are indispensable. Serologic and Roentgen examinations may be necessary.

4. Guarded, Indirect Psychosomatic Inventory:

This is last but not least in the investigation. Such an inventory is distinctly the responsibility of the otologist and should not be shifted to the psychiatrist unless major psychiatric problems are uncovered and demand major psychiatric help. In this inventory it is important not to suggest negative facts, such as insanity, deafness, tumor, etc. It is important to allow the patient to tell his own story and mention his own specific anxiety states and fears, without prompting.

Such an approach to the patient with tinnitus will usually yield a reasonably accurate etiologic diagnosis and will probably establish the proper psychosomatic weight carried by this particular patient-tinnitus combination.

VI. MANAGEMENT OF THE PATIENT WITH TINNITUS:

The goal in the management of the patient with tinnitus is, therefore, a dual one. The first goal in management is, of course, specific therapy wherever and whenever possible. The second goal is the attempt to change an uncompensated tinnitus into a compensated tinnitus in order to make the tinnitus an acceptable sensory phenomenon and detract from its psychosomatic load on the patient.

A. Specific Therapy:

The specific therapy to be applied in the patient with tinnitus depends entirely, of course, upon the anatomic location and the pathologic change. Inasmuch as this aspect entails the entire scope of otologic diseases, it is not within the sphere of the present discussion to enter upon this subject even lightly. We will all agree that in many instances specific therapy is known and successful. We will as easily and quickly agree that in many instances specific therapy is either unknown or impossible at the present time. Suffice it to say that there are numerous instances where specific therapy is applicable. It can be shown, for example, that tinnitus due to serous tympanic effusions may be quickly relieved by appropriate treatment. Tinnitus due to toxic neuropathies produced by drugs may in some instances be relieved by withdrawal of the offending drugs. Tinnitus due to otosclerosis is occasionally improved following fenestration surgery and occasionally improved with a hearing aid. Tinnitus due to acute acoustic trauma is usually relieved by the removal of the offending noise source. There are numerous other illustrations of the reversibility of the symptom of tinnitus. Time does not allow complete discussion.

B. Psychotherapy:

In most instances simple psychotherapy by the otologist is far more effective than such therapy in the hands of the psychiatrist. Occasionally such therapy in the hands of the psychiatrist might actually be detrimental where the problem is not a major psychiatric disturbance. It is only in rare instances that expert psychiatric help should be employed in the patient with tinnitus.

It is of the utmost importance to determine why the patient sought otologic advice at this particlar time.

a. Was it because of the actual intensity of the tinnitus? This is a very rare cause.

b. Was it because of anxiety regarding the meaning of this tinnitus and regarding its possible future repercussions? This is a very common cause for a patient seeking otologic advice.

1. Technique: A thorough examination is obviously important for two reasons: *a.* for actual diagnostic necessity, and *b.* to convince the patient of the thoroughness and validity of the otologist's reassurance.

2. It is necessary to explain to the patient what tinnitus represents, namely, that it is a real and not an imagined thing, that it is not an *illusion nor a hallucination or delusion. It is an auditory paresthesia.* In this regard it is frequently valuable to give the patient the example of the phantom limb with neuron memory and to dismiss from the patient's mind the mistaken and concealed fear that the tinnitus is a sign of mental deterioration.

3. Be sure to explain to the patient *"what tinnitus is not."* Reassure all his doubts, guardedly but firmly.

4. Be optimistic regarding the duration and the severity of tinnitus, remembering that in most cases of tinnitus the intensity will gradually decrease with the passage of time.

VII. SUMMARY:

The successful management of the patient with tinnitus depends, therefore, upon the establishment of an etiologic diagnosis and in the assessment of the psychosomatic weighting factor. This should make it possible to attain the final desired goal, namely, the conversion of an uncompensated tinnitus into a psychologically compensated tinnitus. Any management of the patient with tinnitus which does not take into consideration these two aspects will usually be ineffective. Any management which is based upon a single panacea for the treatment of a symptom and not a disease will result in failure.

INTRACRANIAL LESIONS SIMULATING FRONTAL SINUS INVOLVEMENT.*

CLARENCE W. ENGLER, M.D., and SPENCER BRADEN, M.D.,
Cleveland, Ohio.

Frontal headache is not infrequently the main symptom, and, in fact, sometimes the only symptom of an intracranial lesion. For this reason, frontal sinus disease is erroneously suspected in some of these cases, and they are referred to the rhinologist for treatment. Even though there may be a coincidental frontal sinusitis, the rhinologist should not overlook the possibility of referred pain, due to some type of intracranial lesion. While there are a great number of recognized conditions which may cause pain to be referred to the frontal area, the scope of this paper necessarily limits the discussion to some of the more tangible and demonstrable anatomic lesions.

To have a proper conception of the manner in which pain from an intracranial lesion may be referred to the frontal region, it is necessary that one have an understanding of the pain-sensitive structures of the head, the mechanism by which pain is produced and the neural pathways along which the painful impulses are transmitted. For our knowledge of these factors we are indebted to the very extensive and exhaustive work of Wolff and his various associates at the Cornell Medical Center. The review presented here is drawn largely from his many publications and also those of other investigators in this field. The material is taken from three principal sources: *1.* Moench's book on "Headache," *2.* the *Proceedings of the Association for Research in Nervous and Mental Diseases* for the year 1943, and *3.* from Wolff's monograph, "Pain."

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*Read at the meeting of the combined Middle and Southern Sections of the American Laryngological, Rhinological and Ootological Society, Inc., Memphis, Tenn., Jan. 16 and 17, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1950.

The pain-sensitive structures of the head are both extracranial and intracranial. The scalp—especially its arteries—is sensitive to pain. This is not true of the veins of the scalp, the cranial bone or the diploe; the periosteum of the skull is only moderately sensitive. Briefly, the intracranial pain-sensitive structures include the dural arteries, the basal parts of the cerebral arteries, parts of the dura at the base of the skull, along the olfactory groove and at the roof of the orbit, the great venous sinuses and their venous tributaries from the surface of the brain, the Vth, IXth and Xth cranial nerves and the upper three cervical nerves. The brain tissue itself, the greater part of the dura and the pia arachnoid, the lining membrane of the ventricles and the choroid plexus are insensitive to pain.

According to Wolff, a line drawn vertically over the top of the head from ear to ear divides the pain-sensitive structures into a supratentorial group and an infratentorial group. Stimulation of any of the pain-sensitive structures anterior to this line, *i.e.*, the supratentorial group, will produce pain in various regions in the anterior portion of the head, *e.g.*, the eye and the forehead. Stimulation of the pain-sensitive structures behind this line, *i.e.*, the infratentorial group, will produce pain in various regions in the posterior portion of the head, *e.g.*, the occiput and behind the ear. This is explained by the fact that the sensory division of the Vth cranial nerve supplies the superior surface of the tentorium and all the pain-sensitive structures above it, while the pain produced by stimulation of the infratentorial group is mediated by the IXth and Xth cranial nerves and the upper three cervical nerves.

Wolff has outlined six basic mechanisms by which headache may be produced from intracranial disturbances. The most important of these have to do with alterations in the cranial blood vessels, whether they be displacement of the large venous sinuses or traction on their venous tributaries, or distension and dilatation of or traction on the intracranial arteries. Less important are the inflammatory changes in and about the pain-sensitive structures of the head and direct pressure by tumors on the cranial and cervical nerves. In certain cases, more than one of these mechanisms may be in operation and perhaps more than one pain-sensitive structure may be stimulated. Pain resulting from intracranial lesions is most always referred.

Peripheral painful stimuli initiated from the areas under consideration, on reaching the thalamus, are carried to the sensory cortex of the brain, the postcentral gyrus, where the sensation of pain is appreciated, by way of the thalamocortical radiation.

ILLUSTRATIVE CASES.

It has been stated aptly by Bailey that the first step toward establishing a diagnosis of intracranial tumor is that the possibility of its presence should occur to the physician. This statement can be enlarged to apply to other intracranial lesions.

The following cases are examples of a variety of cranial and intracranial conditions which produced pain in the frontal region.

Case 1: A white male, 55, had had recurring nasal polypi since he was of school age, which had been repeatedly removed by surgery. Eventually there was perforation of the cribriform plate. His presenting complaint, aside from nasal polypi, was severe frontal pain, especially on the right side. Later the patient had coma and minor signs in the extremities of the left side. The lesion responsible was an abscess of the right frontal lobe of the brain.

Case 2: A white female, 53, had severe pain in the right frontal region for two weeks, followed by right IIIrd nerve palsy. The lesion responsible for these symptoms was an aneurysm of the right internal carotid artery.

Case 3: A white female, 36, had had chronic recurring frontal headache since childhood. Later there was a Jacksonian convulsive component, with recent paralysis of the arm and leg. The cranial lesion was a right parasagittal cortical hemangioma.

Case 4: A white male, 56, had recently suffered from unexplained diminution of vision in the right eye and recurring pain in the right frontal region. He had a calcified aneurysm of the right internal carotid artery.

Case 5: A white male, 30, had suffered from right frontal pain extending onto the right side of his face, which had been of abrupt onset. An involvement of the Vth nerve at the Gasserian ganglion was erroneously suspected. The actual finding was an arteriovenous aneurysm of the right parietal cortex.

Case 6: A Negro, 35, had an aneurysm of the right middle cerebral artery. The initial symptom was recurring episodes of right frontal pain, with recent rupture and intracortical hemorrhage.

Case 7: A white female, 25, had had recurring episodes of frontal pain for 10 years. Recently there had been a subarachnoid hemorrhage, with recovery, followed by partial motor aphasia and right hemiplegia. The lesion responsible was a cavernous hemangioma of the left temporal lobe.

Case 8: A white female, 47, had vague right frontal discomfort followed by swelling of the right upper lid and eventual right proptosis, resulting from an osteoma of the lateral wing of the right sphenoid bone and the lateral wall of the orbit.

Case 9: A white female, 27, had vague unexplained right frontal pain for nine years with an uncorrectable visual disturbance of the right eye. She had had a recent episode of subarachnoid hemorrhage, with recovery, but persistence of pain and visual disturbance. More recently, there had been sensory Jacksonian attacks, involving the left extremities. The brain lesion was an astrocytoma of the right frontal and temporal lobes.

Case 10: A white male, 60, had a meningioma of the left frontal lobe with skull changes of 20 years' duration. For four years, he had suffered from left frontal pain and headache. Recently there had been a convulsion with coma and aphasia.

Case 11: A white male, 67, had had a swelling on the back of his head for four years and bifrontal pain for several months. The lesion responsible was an extradural meningioma situated at the midline in the posterior parietal portion.

Case 12: A white male, 66, had a spongioblastoma multiforme of the right temporal and parietal lobes which produced bifrontal pain and visual disturbances with demonstrable homonomous hemanopsia.

Case 13: A white female, 32, had right occipital and right frontal pain with extension onto the right side of the face for 22 months, for which an hysterical origin had been suspected. Eventually, obvious cerebellar signs led to the discovery of a meningioma of the right cerebellum.

SUMMARY AND CONCLUSIONS.

1. The situation of pain-sensitive areas, intracranial and extracranial, and pathways concerned in the transmission of pain to the frontal region from intracranial lesions are reviewed and discussed.

2. Tangible, demonstrable anatomic lesions producing reference of pain to the frontal area are reported.

3. The frequency of frontal pain resulting from an intracranial lesion demands consideration of a variety of cranial or intracranial conditions other than frontal sinus disease, as the possible causative agent.

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NATIONAL ASSEMBLY OF SURGEONS OF MEXICO.

Mexico City.—The meeting of the Plastic Surgery Section of the Ninth National Assembly of Surgeons will be held Nov. 19-25, 1950, at the Hospital Juarez in Mexico City.

The meeting of the American Society of Plastic and Reconstructive Surgery will be held in Mexico City, Nov. 27-29, 1950, immediately after the work of the Plastic Surgery Section of the Ninth National Assembly of Surgeons. The management of this meeting will be in charge of the American directors. Each group may execute their work according to their usual procedure.

We hope to include the meeting of the Latin American Society of Surgeons and thus make this the First International Congress of Plastic Surgery in Mexico.

For further information address the Secretary, Dr. Alfonso Duenas de la Torre.

BLOOD DYSCRASIA IN OTOLARYNGOLOGY.[*]

H. JAMES HARA, M.D.,[†]
Los Angeles, Calif.

The initial clinical symptoms of acute hematological disturbance, not infrequently, appear in the field of otolaryngology. Many patients with insidious chronic progressive blood disorder are not aware of this condition until the occurrence of protracted nasal hemorrhage, sudden vertigo and deafness or development of oral lesions which prompts them to seek the services of otolaryngologists.

The present study is based on the following cases of blood dyscrasia observed at the White Memorial Hospital, Los Angeles, from 1939 to 1949. These cases originated either *1.* in my own practice, *2.* in the outpatient department and later admitted to the hospital, or *3.* in the practice of my colleagues in other departments of the hospital in which otolaryngological consultations were sought.

1. Hemophilia		4
2. Polycythemia Vera		8
3. Infectious Mononucleosis		30
4. Agranulocytosis		8
5. Purpura Hemorrhagica		22
6. Pseudohemophilia		2
7. Leucemias:		76
Lymphatic	41	
Myelogenous	29	
Monocytic	6	
8. Telangiectasia Hemorrhagica		9
		159

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[*] Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., Jan. 28, 1950.

[†] From the Department of Otolaryngology, College of Medical Evangelists, Los Angles, Calif.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

1. HEMOPHILIA.

Hemophilia is an hereditary blood disorder. The bleeders are limited to males, but females of the family transmit the disease to the male offspring. Its incidence follows the Mendelian law in that a recessive sex character is related to a single male chromosome. A complete review of the subject was given by Davidson and his associates.[1] According to the newer concept, the hemophiliac's blood lacks a clot-forming element in its plasma.[2] The blood of the hemophiliac shows normal distribution of cellular elements. His bleeding time is normal, but coagulation time is prolonged. There is no abnormality of the capillaries. Of the four hemophiliacs in this series, all were under 25 years. Each gave a history of a lifelong tendency to bruise and slow, oozing type of bleeding after the slightest cut. The majority of the hemophiliacs bleed to death in childhood.

Five-year-old Kenneth had the history of epistaxis since three years of age and could not blow his nose without producing nasal bleeding. This child fell from a swing in his back yard and died from intracranial hemorrhage on the eighth day following the accident. The remaining three cases were ages 21, 23 and 25, with numerous episodes of bleeding from the mouth, lips and tongue. Each had bleeding when their teeth erupted and received medical attention. One had died in another hospital. The third could not be traced. The fourth patient, aged 23, is a semi-invalid and became a morphine habitue on account of the panhemarthrosis.

No organs are spared from hemorrhage. Hemarthrosis and hematuria are present in 90 per cent of cases.[3] Epistaxis is universal.[4] Intralingual and paralaryngeal hemorrhage is a serious complication. Three of seven received a tracheotomy.[5,6] Bleeding into the ear masquerades as acute mastoiditis. Diagnosis should not be difficult. In one out of three cases heredity may not be established.[7] Craddock and his associates[8] and Shea[9] warn that the clotting time determination may be misleading in the hemophiliac, since it takes but a small amount of thrombin in clotting when a venopuncture is made. A disastrous hemorrhage may occur when a larger vessel is severed in the same individual. The amount of prothrombin remaining in the plasma after clotting time is a much more reliable test for hemophiliacs.

Having found the missing blood factor, this deficiency is now supplied by the antihemophilic globulin (Fraction I of Cohn) or by fresh normal plasma.[10-14] Injections of whole blood, human and animal globulin and human placental extract and thrombin preparations may also be used to tide the patient over a critical period. Much can be done if injury to hemophiliacs is avoided and proper preoperative precaution is taken. The preservation of injectable veins is an important item in the management of every hemophiliac. Nasal packing for epistaxis is dangerous. A tracheotomy set should be in easy reach when pharyngolaryngeal hematoma is suspected.

2. POLYCYTHEMIA VERA (VASQUEZ-OSLER DISEASE).

This is a slowly progressive, chronic disease of the bone marrow with many remissions and fatal termination. Its onset is insidious. Most patients date the inception of their disease back more than 10 years.[1,2] It is a disease of past middle life, some cases being familial. The highest incidence is found among the Hebrew race.[3] Under some unknown stimuli there is a striking erythroblastic activity of the bone marrow, the red cells sometimes exceeding 8,000,000 per cm. This unrestrained production of red cells is also accompanied in some cases by an increased production of immature white cells and platelets, the prognosis of which is extremely poor.[1] The proportion of cells to plasma is greatly increased in typical cases.[4] This increases the viscosity of blood which tends to elevate the blood pressure, predisposes to thrombosis and gives the blood a grossly dark color.[5]

Frequent epistaxis, headaches, postural vertigo, tinnitus, fullness in the head, mental confusion, dyspnea and cardiac embarrassment are the chief symptoms. Bleeding occurs from congestion rather than from hemorrhagic tendency. Hemorrhage and anemia occur late in the course of the disease. The physical appearance of patients suffering from polycythemia is most striking. "Red as roses in summer and blue as indigo in winter" is the description given by Osler.[6] The eyes are bloodshot. The oral mucosa is deeply cherry. The tongue is swollen, beefsteak in color. The pharynx presents a diffuse, fiery red appearance.

There were two males and six females in this series. The youngest was 35; the oldest, 70; the majority being past 60 years of age. The following case is typical of this malady:

Mrs. E. C., aged 49. For the past year and a half before her admission to the hospital, she had numbness, substernal oppression, headaches and repeated epistaxis. During the same period she had several teeth extracted, and on one occasion bleeding of the gums continued for four days. Four months before her admission to the hospital she consulted her family physician for severe headaches and dyspnea and found that her red cell count was 7,000,000 per cm.; hemoglobin, 126 per cent. Profuse epistaxis occurred frequently. Later she was admitted to the hospital for diagnosis and remained for a week. Two venesections gave her only temporary relief.

The treatment is palliative. Radioactive phosphorus is now most popular.[7-10] X-ray has been ineffective.[11] Periodic venesections, acetyl pheny hydrazine, nitrogen mustard,[12] Fowler's solution and a diet low in iron[13-15] offer the best relief and prolong the life of the patient.

3. INFECTIOUS MONONUCLEOSIS: (INFECTIOUS LYMPHADENOSIS).

Infectious mononucleosis is a self-limited disease, probably of virus origin, occurring usually in sporadic and at times epidemic form among children and young adults (see Fig. 1). No race is spared, but Negroes are more susceptible.[1] Incubation period is seven to 15 days.

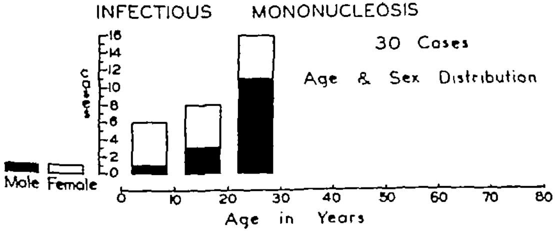

Fig. 1. Infectious mononucleosis is a disease of children and young adults. The sex is evenly divided.

Two major types are generally recognized: the pharyngeal and lymphatic varieties.[2,3] Some subdivide them as glandular, anginose and febrile, but they may merge into each other.[4-11] In the pharyngeal type the most excruciating form of sore throat is the outstanding symptom. Following the initial period of malaise, headache, fever of a few days to a week, the patient complains of acute pharyngitis. The tonsils, if present, are invariably enlarged and often coated with grayish, odorless exudate. The culture may show Vincent's organisms, various forms of streptococcus and staphylococcus. In rare instances the throat presents the clinical picture of diphtheria. Laryngeal stenosis may require a tracheotomy.[12] In the severe form there is always profound toxemia, simulating typhoid fever or an atypical pneumonia.[11,13] In the milder forms the patient may be afebrile practically throughout the entire course of the disease. The only clinical sign may be an enlarged cervical gland and a slight evidence of upper respiratory infection.

In the lymphatic type the adenopathy is the chief complaint. The cervical nodes may become tender and enlarged. At times, one must differentiate the condition from mumps. The glands of the axilla and the groin are also enlarged. Splenomegaly is present in two out of three cases. The cough may become troublesome due to enlarged mediastinal lymph nodes. Bizarre neurolgical manifestations have been recorded.[13,14] Epistaxis and hematuria are not uncommon.[15] Purpura[16,17] and jaundice may be a complication.[18-22] The disease may last a number of years.[23] So protean are the clinical manifestations of the disease it often taxes one's diagnostic acumen.

In a series of 30 cases, a correct diagnosis of mononucleosis was recorded on admission in only eight instances. Another group of seven patients was admitted without diagnosis. In the others admission diagnoses were acute pharyngitis, streptococcus sore throat, acute tonsillitis, Vincent's angina, hypertrophic adenoids, mumps, diphtheria, poliomyelitis, leucemia, pyelitis, jaundice, rheumatic fever, influenza, pneumonia and acute appendicitis. The latter diagnosis was derived probably from enlarged mesenteric lymph nodes.

The condition usually persists for several weeks with a tendency for remission and recurrence. Diagnosis is made from *1.* the clinical picture of headache, asthenia, fever of 100° to 104° F., sore throat with or without ulceration and enlarged, tender adenopathy; *2.* the presence of a positive heterophile antibody reaction (Paull-Bunnell's) in a titer 1:16 or higher, and *3.* the recognition of atypical leucocytoid lymphocytes in the circulating blood.[1,4]

During the initial stage all three of these syndromes may not be present; but if two are present in the course of the disease in the patients under 30 years of age, in a community in which upon inquiry other similar age groups have had a similar condition, the diagnosis is probably correct. The positive heterophile reaction is obtained most frequently during the second and third weeks of the disease. In a typical case there is an initial leucocytosis or normal count followed by a drop to leucopenia with a rising mononucleosis. When the diagnosis is in doubt, repeated smears and blood counts are advisable. The prognosis is good in uncomplicated cases. There are now on record nine cases of ruptured spleen resulting from this disease.[14-24] Two deaths occurred from infectious polyneuronitis, one each from nasopharyngeal hemorrhage and laryngeal edema.[13]

Diagnosis may be extremely difficult as shown in the case cited below:

A. K., a 24-year-old, white medical student, was first seen Feb. 3, 1949. His chief complaints were nasal obstruction, frontal headache, fever, 102° F. Physical signs were that of acute rhinitis, hyperplasia of the pharyngeal lymphoid tissue and cervical and submaxillary adenopathy. Because of marked prostration and high fever, he was admitted to the hospital with an admission diagnosis of influenza. The first hematological study revealed nothing of abnormal nature (see Fig. 2). The blood study and heterophile reaction on the tenth day of illness established the diagnosis. The temperature fluctuated during the first week and subsided by lysis the following week (see Fig. 3).

The treatment of infectious mononucleosis has been largely symptomatic. Recently daily intramuscular injections of serum globulin in doses of 10 cc. have been tried and found to be specific for this condition.[27]

INFECTIOUS MONONUCLEOSIS

Patient: A. K

Date:	Feb. 4	Feb. 6	Feb 10	Feb. 11	Feb. 12	Feb. 14
Hemoglobin	96%					
Erythrocytes	4490000	4900000	4800000	5020000	4870000	4870000
Leukocytes	7900	3650	8200	10100	9900	9450
Lymphocytes	23%	37%	23%	12%	10%	12%
Monocytes	3%	7%				
Eosinophils						
Basophils						
Neutrophil Band Cells	74%	50%	31%	34%	18%	23%
Promyelocytes		2%				
Myeloblasts		2%				
Prolymphocytes			46%	53%	72%	64%

Fig. 2. Typical hemogram in infectious mononucleosis. Patient was a 24-year-old white medical student. On the fourth day of illness on Feb. 4, 1949, his cell count was normal. Feb. 6, there was leucopenia and a slight neutropenia. On the tenth day and thereafter there was prolymphocytosis until on his twentieth day of illness when the blood count became normal.

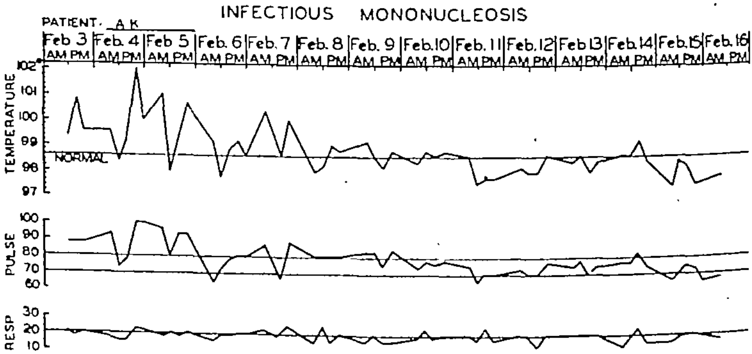

Fig. 3. The initial rise in temperature, pulse and respiration rate was followed by a gradual decline.

4. AGRANULOCYTOSIS.

Since Schultz's first paper on agranulocytosis appeared in 1922[1] interest in this disease has been unabated. During the past 10 years the *Quarterly Cumulative Index Medicus* lists more than 740 references on this subject. The present concept of this condition may be thus summarized:[1-8]

1. The disease is an idiosyncrasy to some drug, the dosage-time factor of which has no relation to the intensity of symptoms. Women past 25 years of age are particularly susceptible. *2.* The most frequent offenders are found among the derivatives of the benzene ring, notable of which is aminopyrine. The next in order are dinitrophenal and sulfonamides. Among other drugs are thiouracil, barbiturate, various quinine derivatives, bismuth, gold, mapharsen, arsenicals, dilantone, pyribenzamine, tridione and hydantal. *3.* It also develops secondary to hyperplastic processes in pernicious anemia, leucemia, purpura, allergic reaction and acute infection. *4.* The characteristic hematologic change is brought about by suppression of maturation of granulocytes during the myeloblastic stage. The total count may not exceed 1,000 per cm. *5.* There is no alteration in red cell and platelets counts in a typical case, a factor which accounts for lack of hemorrhage. *6.* The sepsis is secondary to leucopenia and when untreated the patient succumbs to overwhelming sepsis within 36 to 72 hours of apparent onset.

The most characteristic feature of the disease is the rapidity of the course, profound toxemia and striking response to the drug withdrawal combined with antibacterial therapy in favorable cases. The disease is ushered in with alternate chills and high fever, marked prostration, malaise, headache, cervical adenopathy and necrotizing mucous membrane of the ear, nose, pharynx and oral cavity.[9,10] The soft palate, uvula and the pillars of the tonsils are extremely reddened and eroded. The process may extend to the larynx, the obstruction of which requires a tracheotomy.[11-13] The blood picture is so typical that little difficulty is to be expected in establishing a diagnosis. In the initial stage there is asymptomatic leucopenia and neutropenia. The lymphocytes consist nearly 90 per cent of the total white counts. There is no known sensitization test to determine one's idiosyncrasy, thus every trace of drug must be eliminated from the patient, and in practice this is not always easy. At times the patient's own secretiveness defeats this very object. A young married man consulted me for the treatment of acute pharyngitis, which later was diagnosed as an agranulocytic angina. Unbeknown to me, the patient was in the habit of self-drugging off and on for a period of months with a fairly large dose of sulfonamide for a Neisserian infection which he acquired from extramarital source. On the initial visit the patient denied the drug habit. Confronted with a correct diagnosis based on hematologic study and clinical picture, the patient finally disclosed the history of the urethral infection, subsequent self-treatments and development of the recent oral lesions for which he sought my services.

The prognosis is excellent when aggressive therapy is instituted. Pentnucleotide, transfusions, pyridoxin and folic acid have enjoyed popularity; however, penicillin appears to be specific. The most important feature is its prevention,[14] and the instant withdrawal of the causative medication when diagnosed.

5. THROMBOCYTOPENIC PURPURA.

The simplest yet most comprehensive classification of purpura is given by Fowler:[1]

I. Thrombocytopenic Purpura:

- *A.* Idiopathic or essential.
- *B.* Secondary purpura associated with some other recognizable disease, intoxication and infections.

II. Nonthrombocytopenic Purpura:

- *A.* Idiopathic.
  - *1.* Schönlein-Henoch or allergic purpura.
  - *2.* Purpura simplex.
- *B.* Symptomatic purpura.

This paper is limited to the discussion of thrombocytopenic purpura, or Werlhof's disease. Pathogenesis of this condition has been thoroughly studied by competent investigators.[1-10] There were 22 cases in this series. All of the patients had in common the tendency to an abnormal bleeding into the skin and mucous membrane, a marked reduction in the number of platelets in the circulating blood, prolonged bleeding time, a defective clot retraction, a positive tourniquet test, and normal coagulation time. The disease is most frequently seen in children and youth; women suffer twice as often as men. It is rare in the Negro.[11] There is a distinct hereditary tendency. The cause of hemorrhage is not known, but is thought to be the combination of thrombocytopenia and capillary anomaly.[12,13]

Quick[14] has explained this platelet-capillary phenomena on the basis of histamine activity[*] and it appears to be gaining favor.[15] The course of the disease is both acute and chronic. The acute form lasts two to several weeks. It may terminate in complete restitution or in death from hemorrhage or aplastic anemia due to repeated blood loss. In the chronic form the disease shows a tendency to frequent remissions. The episodes of bleeding may last from a few days to a few weeks. The number of platelets may attain normal level during the symptomless intervals.

Epistaxis is one of the chief initial symptoms that brings the patient to a clinician. It is not unusual for the illness to appear following an acute upper respiratory infection. Without hematological investigation a diagnosis may be difficult if the cutaneous lesions are few or absent, as in the initial stage of most of the cases.[10] In 15 of the series of 22 cases, the disease was precipitated with epistaxis. A history of a tendency to bruise on slight trauma, an episode of hemorrhage from the nose, throat or vagina should arouse suspicion, or at least raise a question.

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[*] Histamine causes capillary dilatation. One of the functions of platelets is to remove excess histamine. In so doing, platelets are rendered more susceptible to agglutination and lysis, resulting in thrombocytopenia and purpura. This concept of allergic origin explains both the thrombocytopenic and nonthrombocytopenic purpura.

At the age of 12, a Mexican boy had a tonsillectomy in another hospital. This was followed by a prolonged, oozing type of bleeding, typical of this condition. He received eight transfusions before the bleeding was controlled. He has had a tendency to bruise all his life and frequent epistaxis since two years of age, but this vital point in the clinical history was apparently overlooked in the preoperative examination.

Pregnancy adds hazards to the condition.[17] Two of my three cases spontaneously aborted and died from massive intrauterine hemorrhage; upon the third a therapeutic abortion was performed. All three had a daily epistaxis during the period of their hospitalization.

Bleeding from gums, mouth and throat are the next frequent symptoms observed. A 46-year-old male had his teeth extracted. This was followed by protracted bleeding. His only son had died following tonsillectomy and adenoidectomy in another hospital from uncontrollable bleeding, in spite of repeated transfusions.

Prognosis is generally poor in the acute form, particularly among children under 10 years of age. Death may occur in a few hours following the initial symptoms.[17,18] In the presence of shock and massive bleeding, the blood loss must be immediately replenished. X-ray and urethane have been the treatment of choice. For chronic cases, a small quantity (100 to 300 cc.) of repeated transfusions at two to three-day intervals have been recommended on the theory of stimulating the blood formation and to supply platelets. A splenectomy in chronic cases offers the best outlook. There were four patients in this series who submitted to this operative procedure. Three are living and well for seven to 10 years following the operation. One died from other causes.

6. PSEUDOHEMOPHILIA.

Pseudohemophilia (Willebrand's disease) is a rare hereditary blood dyscrasia which appears in both male and female and is transmitted by both. Unlike thrombocytopenic purpura, with which the condition at times may be confused, there is no deficiency in the number of platelets in the circulating blood. There is no splenomegally; neither is there demonstrable defect in the clotting time nor in clot retraction. The distinct feature of the condition is a prolonged bleeding time.

Willebrand[1] originally suggested that the condition was due to anomaly of the capillaries, and his suspicion was confirmed by Macfarlane, who has shown that in pseudohemophilia there is failure of the capillary to retract upon injury.[2,7]

A number of excellent clinical reports have appeared, both in the American and German literature, under such titles as Familial Hemorrhagic Condition, Atypical Pathologic Hemorrhage, Konstitutionelle Thrombopathie, Familial Purpura, and Hereditary Pseudohemophilia.[8-11] Hewlett and Haden[12] collected 73 cases from the literature and added four cases of their own. The present series makes the total of 79 that have been recorded.

Differentiation must be made from thrombocytopenic purpura.[13,14] There is no specific treatment. Transfusion restores the blood volume, and combats a secondary anemia. Of the two patients in this report, one was a 60-year-old woman who had the history of lifelong, frequent epistaxis and ecchymosis on the slightest trauma. Her son also had a similar affection. On one occasion she had a large sublingual hematoma that made it difficult for her to talk or swallow. As she grows older, these symptoms are becoming increasingly less severe. The second was a 28-year-old woman who has had repeated spontaneous bleedings from her nose and gums. Her mother, a sister and her mother's two brothers also were similarly affected.

7. LEUCEMIA.

Leucemia is a fatal disease of irreversible, proliferative process affecting one of three types of white cells and their immature precursors in the blood-forming organs. They are lymphatic, myelogenous and monocytic leucemias. The disease appears in acute, subacute and chronic forms.[1-7] In the course of the disease there is an increase of white cells, 50,000 leucocytes per cm. being generally accepted as a dividing line. In so-called aleucemic form, the leucocytes may not exceed 10,000 with the normal differential count. The disease is apparently on the increase.[8,9] Since 1940 the annual toll from this condition exceeds 5,000 in the United States.

The acute lymphatic leucemia occurs usually in children and youth. The acute myelogenous type occurs most frequently after 50 years of age. The acute monocytic leucemia occurs after 30 years of age. In all acute leucemias the onset is abrupt, not unlike acute infection. There is high fever, prostration, malaise, tonsillitis and headache. Cervical adenitis is a prominent feature in acute lymphatic leucemia. There is progressive anemia and its associated symptoms. Bleeding from the nose, the gums, mouth and throat is frequent and should arouse the suspicion of the clinician. Ulceromembranous stomatitis occurs in 40 to 50 per cent of cases based upon a large series of acute leucemias.[10-13] The disease runs a rapid, galloping course. In fulminating cases, the disease terminates in a few days to two or three weeks. A spontaneous remission in acute leucemia has been recorded.[14]

Of the 76 cases of leucemias in this series, 25 were classified as chronic. These chronic cases ran a milder course and occurred in older individuals. There are several features which were common to all chronic leucemias: *1.* Progressive weakness, fatiguability, pallor, dyspnea and palpitation, and secondary anemia; *2.* splenomegally, especially in chronic myelogenous forms; *3.* purpura of skin and mucous membrane; *4.* marked tendency toward remission and recrudescence; *5.* slow but steady downward course, terminating in death in two to three years in chronic myelogenous leucemia, five to six years in chronic lymphatic leucemia, and one to two and one-half years in monocytic leucemia.

The most common leucemic symptom which is of interest to the otolaryngologist is epistaxis.[15] Leucemic cutis of the nose is rare.[16] Intranasal leucemic infiltration may cause obstruction.[17,18] Protracted nasal hemorrhage may be the initial symptom that causes the suspicion of leucemia.

Intraoral lesions of leucemia appear also as hemorrhage, infiltration and ulceration.[12,13,15] The infiltration may be local or diffuse. Ulceration of the surface follows, such as an invasion from thrombosis. Hemorrhage following extraction of teeth is frequent. Sometimes the involved gums undergo ulceration with no tendency to heal and exposing necrotic bone deep in the tooth socket, covered with foul, grayish exudate, and sometimes the gums present the classical picture of Vincent's angina. Leucemic infiltration of the tonsils may simulate peritonsillitis.[19] Usually one tonsil is larger than its mate. Pain is severe, but an incision produces no pus. The infiltration may extend to the larynx, requiring a tracheotomy. A safe rule is to suspect leucemia in unilateral enlargement of tonsils until proved otherwise.

The leucemic disturbance of the ear is hemorrhage into the external and middle ears and the labyrinth, causing impairment of hearing, vertigo and tinnitus.[20] Leucemic infiltration may produce the syndrome observed in Ménière's disease.[21] If the patient survives the hemorrhagic episode, there is a new formation of the connective tissue within the labyrinth, constituting "otitis interna ossificans."[21] Paralysis of the VIIth and VIIIth cranial nerves is not rare from this source.[22]

The following otolaryngologic history was recorded on admission of 76 cases in this series:

Cervical adenopathy	29
Epistaxis	17
Dyspnea and palpitation	17
Ulcerative stomatitis	15
Bleeding gums	14
Retinal hemorrhage	8
Deafness	7
Otitis media	5
Tinnitus and vertigo	3
Hemorrhage in ear	2

The treatment of leucemia has been unsatisfactory. Those of proven palliative value are: Irradiation with X-ray,[22-25] radioactive isotope, such as radioactive phosphorus (P32),[26-30] blood transfusion,[31-35] mustard gas,[36,37] and urethane.[38,39] For acute leucemias aminopterin[40] has been found to be helpful.

8. HEREDITARY HEMORRHAGIC TELANGIECTASIA. [RENDU-OSLER-WEBER DISEASE.]

This is an hereditary blood disorder involving both sexes, the lesions appearing in the mucous membrane and the skin as ruby-red telangiectasia. There is no disturbance either in plasma, cells or in their chemical behavior. The primary site of the lesion is in the anomalies of the vascular walls.[1-10] Stock[11] estimated, in 1944, that from a number of reported cases there were probably 175 affected families involving 1,000 members. Mortality ranges from 4 to 6 per cent.[8,12]

The M. family (see Fig. 4): The bleeder in the first generation and another in the second are dead from unknown causes not associated with hemorrhagic disorders. Nine were personally examined by me. Three are in other states, but furnished detailed personal history. The oldest is a 75-year-old widow. Her sons, aged 52 and 50, and daughters, aged 51, 45 and 40, show well marked telangiectatic lesions on their lips, tongue, pharynx, cheeks, nasal septum, turbinates and fingertips. Among the offspring in the fourth generation there were two married women, each aged 20, and three girls, aged 22, 17 and 13. These showed incipient pin-sized cutaneous lesions to fairly well formed ruby-red, spider-shaped telangiectasis of the mucous membrane of the nose and oral cavity.

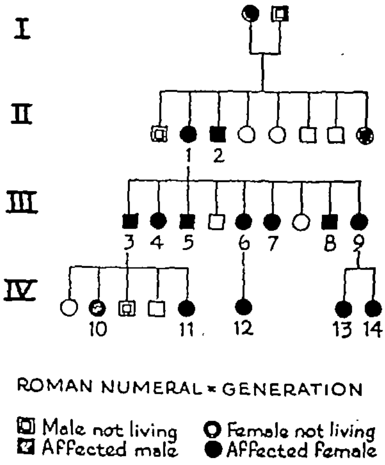

Fig. 4. The bleeder in the first and another in the second generation are dead from causes not associated with the blood disorder. The oldest is a 75-year-old widow. Her sons, aged 52, 50 and 38, and daughters, aged 51, 45, 42 and 40, show well marked telangiectatic lesions. Among the offspring in the fourth generation there were five girls, ages 22-13, with bleeding tendencies.

Epistaxis is the initial symptom. Bleeding begins without any appreciable cause or following nose blowing, sneezing or coughing. Three had developed nasal bleeding before their tenth year. In others, it came on after the thirtieth year. Emotional disturbances play a distinct part in precipitation of epistaxis. Joking, laughing and crying are often predisposing factors. Bleeding may occur in any part of the body. Blood loss may be so insignificant that the patient seeks no medical care or it may be so profuse, occurring at frequent intervals, that well-marked signs of secondary anemia are present. Diagnosis presents no difficulty. There is no known specific remedy. Aside from control of hemorrhage and secondary anemia, oral administration of hesperidian methyl chalcone appears to be helpful.[13]

COMMENTS.

Hemorrhagic disease of the newborn is a pediatric problem and not included in this study. Patients presenting the Plummer-Vinson syndrome (evidences of microcytic, hypochromic anemia, achlorhydria, nutritional disorder, glossitis, cheilitis and splenomegaly) were occasionally seen in the dispensary, but they are not included in this paper. Probably subclinical cases of avitaminosis were overlooked, but patients suffering from scurvy with bleeding from the gums were not encountered.

SUMMARY.

1. One hundred fifty-nine cases of various blood dyscrasia in otolaryngologic practice are reviewed.

2. One cannot place too much importance on a well elicited clinical history, including the tendency for bleeding in the family and patient, a careful search for evidence of nutritional deficiency and any deviation from normal in the hematologic investigation.

3. Repeated blood examinations are sometimes necessary before a diagnosis is made. It is also an indispensable procedure in determination of the therapeutic efficacy and final prognosis.

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THE LOCAL APPLICATION OF "SULFAMYLON" [PARA-(AMINOETHYL)-BENZENE SULFONAMIDE HYDROCHLORIDE] IN OTITIS EXTERNA AND CHRONIC OTITIS MEDIA.*

A Second Report of 164 Infections in 122 Patients.†

J. W. McLaurin, M.D.,
Baton Rouge, La.

Otolaryngologists who practice in the South and Southwest of the United States have come to accept otitis externa as one of their perennial problems. I would not imply that it is a problem which has been solved, but certainly we have come a long way toward its solution. Within the last decade the literature of otitic infections in general and of otitis externa in particular has begun to approach adequacy: comprehensive studies in classification have been made; fungi have been assigned their proper rôle as etiologic factors; the rôle of other micro-organisms has come to be appreciated, and finally, methods of treatment are becoming a good deal more rational. When one examines one's old records, as I recently examined mine, it becomes very clear indeed how much progress has been made.

It has been a source of considerable surprise to me to learn from certain comments on my preliminary publication on the subject of sulfamylon [para-(aminoethyl)-benzene sulfonamide hydrochloride] in the treatment of otitis externa and chronic otitis media[1] that I am credited with recommending this agent in the management of all otitic infections. It is difficult to know how such an impression could arise from a

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*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 16, 1950.

†From the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, and Our Lady of the Lake Sanitarium, Baton Rouge, La.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

paper which bears a title specifically limiting the use of the drug to the two conditions mentioned. Sulfamylon is no panacea and was not proposed as such. It is doubtful that a single agent will ever be found which will cure all varieties of otitic infection. Sulfamylon should certainly not be used for any such purpose. It would not be indicated in the fungoid types of infection, for instance; though it would probably do no harm, it is quite unlikely that it would do any good. It could have tragic consequences if it were employed in chronic otitis media of Lillie's[2] type III or type IV, in which surgery is clearly indicated.

As I related in my first communication on this subject,[1] my attention was attracted to the possible usefulness of sulfamylon in otitic infections by Clark's report on its effectiveness in the prevention of infection after ophthalmic surgery. His published report[3] concerned 84 successive operations after which Bacillus pyocyaneus, formerly an extremely troublesome secondary micro-organism, was completely controlled by the use of sulfamylon in 1 per cent solution for irrigations and moist dressings. Although dressings had to be changed frequently, sometimes for as long as a month after operation, there was not a single instance in the series of any of the reactions experienced under similar circumstances with other sulfonamide preparations.

BACTERIOLOGIC STUDIES.

Sulfamylon, which is derived from benzalamine, differs from sulfanilamide in that a methylene group has been inserted between the benzene ring and the 4-amino group. It is available as a crystalline white substance which is freely soluble in water.

Sulfamylon has the dual advantage of being both effective and innocuous. Its antibacterial activity, Brewer[4] demonstrated, is not inhibited by p-aminobenzoic acid and, therefore, its range of usefulness is wider than that of other sulfa compounds. Brewer's additional studies, which were confirmed by Lawrence,[5] showed that sulfamylon is more effective against aerobes and anaerobes *in vitro* than are comparable concentrations of sulfanilamide and sulfathiazole. Still other investigations showed that solutions ranging in strength from 0.5 to 2 per cent did not harm the conjunctiva of rabbits and that solutions ranging in strength from 1 to 20 per cent did not harm the bladder mucosa. Comparative studies by Howes[6] showed that for local application sulfamylon has a wider range of usefulness than streptomycin, penicillin, parachlorophenol, tyrothricin or zepharin. Its special advantages, as this observer listed them, are that it is rapidly bactericidal, it is not affected by changes in the acidity of the environment, it is active in the presence of pus and blood, and it is relatively nontoxic.

Studies made for me in the laboratories of Our Lady of the Lake Sanitarium in Baton Rouge showed that sulfamylon was consistently effective *in vitro* against the organisms commonly found in otitis externa and chronic otitis media, that is, pseudomonas aeruginosa, staphylococcus aureus, beta hemolytic streptococcus, Klebsiella pneumoniae, alpha hemolytic streptococcus and escherichia coli. Solutions of sulfamylon ranging in strength from 4 per cent down to 0.25 per cent effectively inhibited growth in all of the 36 cultures tested. Additional studies showed that the optimum bactericidal effect was obtained when a 1 per cent solution was kept in contact with the bacteria tested for a five-minute period. This is a useful and practical bit of information: The success of any mode of treatment of otitic infections depends upon the cooperation of the patient and most patients who are not incapacitated by their condition will not submit to inactivity for much more than five minutes at a time.

ROUTINE OF MANAGEMENT.

The present routine of management in otitis externa, chronic otitis media and occasional postoperative infections has been somewhat modified from the routine described in my first communication.[1] I am in full agreement with Fowler[7] that bacteriologic studies are essential in these conditions, but I am in equally full agreement with Senturia[8] that for a variety of reasons they are not uniformly practical in private practice. I should not advise anyone who is just beginning to treat otitis externa and allied conditions to omit them. On the other hand, I am convinced from my own experience that as experience increases, it is frequently possible to determine *from inspection and clinical examination what sort of infection* one is dealing with and that it is entirely justifiable to treat most patients tentatively, on the basis of that impression, with a considerable saving of time and expense. If by the second visit the ear has not shown great improvement under the treatment instituted, cultures are taken at once. In this connection might be mentioned Senturia's recent statement, in his discussion of Fowler's[7] paper on topical applications to the external ear, that the Los Angeles Research Study Club was sponsoring an investigation which, it was hoped, would *produce a method of cytologic examination* which could be carried out in the office to differentiate among the varieties of external otitis. It would be an enormous diagnostic aid if this could be achieved.

The present routine of treatment is as follows:

1. Following history-taking and the usual general and special examinations, a smear and culture are taken if there is any doubt at all concerning the type of infection presented. The ear is then thoroughly cleansed with the tip suction, at first with the smallest size Frazier suction tip, then, near the attic and the tympanic membrane, with a tip devised in my office for this purpose. This tip is malleable and can be bent to reach otherwise inaccessible areas. *Finally, the canal is* irrigated with one ounce of 95 per cent alcohol and is thoroughly dried.

2. If the infection is thought to be fungoid in origin, the fungicide of choice is prescribed. I prefer thymol and cresatin for this purpose. Otherwise, sulfamylon is applied and the patient is shown how to use it at home. Application is simple: The patient lies on the nonaffected side, instills the sulfamylon solution into the ear and permits it to remain for exactly five minutes by the clock. *The ear is then blotted dry* with cotton and a cotton wick is inserted, which is changed often enough to prevent its becoming saturated with secretions from the ear.

3. When the patient returns to the office, usually 48 hours after the first visit, the ear, as a rule, is found greatly improved, probably as the result of the thorough cleansing combined with the action of the sulfamylon. My own belief is that if it were practical for patients to come to the office daily, it would be possible to clear up most cases of otitis externa merely by repeated daily cleansing. It is of interest that almost without exception observers who report good results with various methods of treatment all emphasize the importance of cleansing the canal and drying it thoroughly before any applications are made to it.

If on the second visit the infection is clearly under control, the ear is again cleansed and dried carefully and is dusted with sulfanilamide powder. The patient is instructed to discontinue all medication and to make every effort to keep water out of the ear for three to four weeks. If, however, improvement has not occurred, the initial treatment is repeated and the patient returns to the office at regular intervals, preferably 48-hour intervals, until control is achieved.

4. In cases in which a culture has been made on the first visit or on a subsequent visit (as is routine in cases in which improvement is not promptly apparent), treatment depends upon the results of laboratory examinations. The report usually shows *1.* mixed organisms, with pseudomonas predominating, *2.* mixed organisms with staphylococcus predominating, *3.* streptococcus, with the precise type named, or *4.* B. pyocyaneus. If a pure staphlococcus is reported, or a mixed infection with staphylococcus predominating, a coagulase test is run. If this test is reported negative, the staphylococcus is disregarded and the infection is treated as if it were gram-negative. That is, sulfamylon therapy is continued. If the test is reported positive, attention is directed toward the staphylococcus. Sulfamylon therapy is discontinued and the patient is treated with penicillin intramuscularly and locally (by drops of a solution containing 100,000 units of penicillin per ounce of distilled water), or by furacin.

To reiterate the principles upon which this clinical routine is based: *1.* The drug selected is applicable to the individual case. *2.* Whatever agent is used is applied in a high enough concentration and over a sufficient period of time to control the infection. *3.* Before any application is made to the ear, and as an essential part of the treatment, the ear is carefully cleansed and dried. The drug is thus enabled to reach all parts of the ear and the aural secretion, the pH of which might alter the effectiveness of the drug, is eliminated, at least for the time being. *4.* The drug is applied only long enough to achieve the desired effects. The long continued use of any type of topical application may induce sensitivity. Dermatologists are of the opinion that penicillin cannot be used locally much longer than about five days without the appearance of erythema, vesiculation and other evidences of sensitization. My own feeling is that similar time limits might well be set upon all topical applications. *5.* If bacteriologic studies are made and a gram-positive organism is found, sulfamylon is withheld, for use in the event that the strain proves resistant, and some other form of treatment is employed first.

ANALYSIS OF CASES.

My preliminary report on the use of sulfamylon in external otitis and other otitic infections concerned 141 infections in 108 patients. In the series were 33 cases of external otitis, 24 of chronic otitis media, nine infections secondary to the fenestration operation and seven secondary to tympanomastoidectomy. The 73 cultures made revealed pseudomonas aeruginosa, staphylococcus aureus, beta and alpha hemolytic streptococcus, Klebsiella pneumoniae, and escherichia coli. Sulfamylon was effective in every instance in this series. In 93 cases the infection was controlled by the seventh day and in no case was treatment necessary longer than 14 days. The single reaction in the series was proved to be the result of a local allergy which cleared promptly after sulfamylon was discontinued and an antihistaminic drug was administered.

To my first series of sulfamylon-treated otitic infections I am now adding a second series of 164 infections in 122 patients. Sixty-five patients had otitis externa, 34 chronic otitis media, 12 cavities following tympanomastoidectomy, and 11 cavities following the fenestration operation. The bacteriologic results were in general agreement with those in the first series.

The results of treatment were quite as satisfactory as in the first series. In 80 cases the infection cleared up within two days, in 24 within four days, in 12 within seven days, in three within nine days, and in three within 11 days. The rapidly favorable results — in this second series only six patients paid more than three visits to the office — are advantages which commend this method to physician and patient alike. There were no reactions at all in the second series.

It is true that I have made no formal follow-up of these 230 patients, but I am sure that most of you will understand me when I say that although my practice is exclusively limited to a specialty, it is more or less of the family type, as might be expected in a small city. I have seen most of these patients for other conditions, or heard of them through other patients who were relatives or friends, or have met them as I have gone about my affairs. I feel sure that in the two series there were some recurrences of infection, but I feel equally sure that most of the patients treated were permanently relieved.

COMMENT.

Reports on the use of sulfamylon in otolaryngology are still few. Fox,[9] in 1947, treated 137 patients with rhinosinusitis by this means with such good results that he concluded that this drug was "the most valuable of all chemotherapeutic agents in the local treatment of acute rhinitis and sinusitis." In most cases in the series no other treatment was used, and antrostomy, which had been Fox's practice if antral infections did not clear up after three or four irrigations, was not required in a single case.

The only report of which I am aware which concerns the use of sulfamylon in otitis conditions is by Fowler,[7] in 1948. He used this agent in the treatment of both otitis externa and chronic otitis media whenever pseudomonas was found in the culture. In the first of the 17 cases in his series he applied the drug in 5 per cent concentration in a water-soluble ointment base (carbowax). In later cases he used a 1 per cent solution of methyl cellulose, with a pH adjusted from 4.5 to 6.5.

Diehl and Morris'[10] report deals with a group of patients with otitis media treated with sulfamylon and streptomycin. Fowler[7] used the same combination for mastoid cavities. The addition of streptomycin makes it impossible to evaluate either series from the standpoint of the effectiveness of sulfamylon alone.

I would not, of course, wish to indicate that sulfamylon is the only possible method of treatment in otitis externa and chronic otitis media. There are several other useful methods, in addition to penicillin therapy. My personal experience with dibromsalicylaldehyde (compound 280) was entirely unfavorable and did not in any way duplicate the results reported by Hayes and Hall.[11] I have used furacin, however, with very satisfactory results in coagulase-positive staphylococcic infections, my experience being in accord with the results reported for this form of treatment by Anderson and Steele[12] and by Douglass.[13]

SUMMARY AND CONCLUSIONS.

1. The situation in respect to the management of otitis externa, chronic otitis media, and certain postoperative otitis infections is greatly improved over what it was.

2. Experience suggests that if the ears are carefully cleansed, if care is taken to determine the causative organisms and to use therapeutic agents to which they are sensitive, much of the confusion surrounding the treatment of these conditions can be expected to disappear.

3. It is the duty of otolaryngologists, as Senturia[8] has emphasized, to continue their methods and techniques critically and to confine their therapy to the use of therapeutic agents worthy of trial.

4. This report concerns the treatment with sulfamylon of 164 infections in 122 patients, which are added to the previously reported series of 141 infections in 108 patients. In both series the majority of cases were instances of external otitis and chronic otitis media; but both included a few instances of infection following tympanomastoidectomy and the fenestration operation. In both series the response to sulfamylon therapy, combined with thorough cleansing of the ear, was uniformly satisfactory and almost uniformly rapid. There was one reaction, as the result of local allergy, in the first series but no reactions at all in the second.

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THE LYMPH PUMP MECHANISM OF THE NOSE AND PARANASAL SINUSES.[*][†][‡]

J. MATHEWS ROBISON, M.D.,
Houston, Tex.

The lymph pump mechanism of the nose and paranasal sinuses essentially consists of the anatomic and physiologic adaptation of the sinal mucosa and turbinal tissue to the process of adjusting their tissue pressure to a magnitude which promotes the flow of lymph from their tissue spaces. Other interrelated variables which enter into this mechanism, particularly during pathologic states, are:

1. The relation of the normal and pathologic tissue pressure of the turbinal tissue to the tissue pressure of the sinal mucosa.

2. Lymphatic blockage in the sinal mucosa or in the retropharyngeal lymph nodes.

3. The relation of the colloidal osmotic pressure of the sinal secretion to the colloidal osmotic pressure of the interstitial fluid in the sinal mucosa.

4. The presence of relatively unremovable protein or bacterial products or antigens in the turbinal tissue or sinal mucosa. This factor is important during both purulent and allergic states.

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[*] Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 17, 1950.

[†] This study was aided by a grant from Harry Carothers Wiess.

[‡] From the Department of Otolaryngology of the University of Texas School of Medicine, Galveston, Tex.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 20, 1950.

Information concerning this mechanism has been derived from the following sources:

1. Clinical observation of the relation of nasal stenosis due to increased turgescence of the turbinates to sinal pathology occurring during various types of rhinitis and sinusitis.

2. Study of the histology and pathology of the sinal mucosa with particular reference to its anatomical, physical and functional characteristics.

3. Determination of the colloidal osmotic pressure of the sinal secretion and interstitial fluid in certain types of sinusitis. Examination of these fluids has had the following obvious limitations:

Sinal secretion and interstitial fluid must be present in sufficient quantity and procurable before they can be examined. During acute purulent sinusitis secretion is plentiful and easily obtained and often, as the sinal mucosa becomes very edematous, the interstitial fluid may be aspirated; however, during subacute allergic sinusitis usually it is impossible to obtain secretion or interstitial fluid from the sinal mucosa. Also during chronic purulent sinusitis, although secretion is readily available, the interstitial spaces have become so filled with round cells or fibroblasts that interstitial fluid is not procurable. In instances where specimens are not procurable, opinions of the processes are based upon observations of similar pathology occurring elsewhere, particularly elephantiasis.

The osmotic pressure of the specimens of secretion and tissue fluid examined has been calculated by determining the percentage of albumin and the total protein and using the nomogram of Wells, *et al.*, for computation. The actual numerical values of the protein, osmotic pressure, etc., are not reported in this paper. They will be presented as part of a more technical article.

The physiologic need for the lymph pump mechanism is derived from the requirement of every living cell for food and an excretory mechanism proportioned to the cells' activity. In mammals these requirements are provided by an extravascular tissue fluid derived from the blood plasma which produces a constantly changing pericellular environment for the cells of the tissues. The filtrate derived from the arterial capillary contains water, salts and food (protein) for the body cells. The water, salts and certain diffusable products are absorbed from the extravascular fluid by the venous capillary, leaving the products of cell metabolism, particulate matter, cellular detritus, bacterial products and unused protein to be carried away by the lymphatic system. This arrangement is, in effect, a filtering process which allows molecules of various sizes to find their way from the interstitial spaces to the blood stream by different routes with little or great delay or difficulty. Mammalian lymph flows normally by muscle contraction, massage, active and passive motion of the body, gravity, tissue pressure and pulsation transmitted from the arteries to the lymph vessels. During pathologic states, when the part is immobile, lymph flows by pathologic or increased tissue pressure, gravity and pulsations transmitted from the arteries and dilated arteriovenous anastomoses to the lymph vessels.[1]

In the normal nose and sinuses the tissues are deprived of muscle contraction, active and passive motion and massage to promote lymph flow. Instead, these structures depend upon gravity and normal tissue pressure. When irritation or infection occurs in the nose and sinuses, lymph flow is accelerated by increased or pathologic tissue pressure, gravity and pulsation transmitted from the arteries and dilated arteriovenous anastomoses to the lymph vessels. This limitation of measures available for moving the extravascular fluids in the tissues of the nose and sinuses is reflected in their anatomy. The anatomic adaptation of these structures to their physiologic needs is evident in the molding of the turbinal tissue to irregularities of the nasal cavities and the physical characteristics of the sinal mucosa and turbinates which allows them to alter their tissue pressure rapidly by swelling against their inherent elasticity and contractility and their bony enclosure.

Tissue pressure is gauged by the resistance a tissue offers to dilatation of its interstitial spaces by edema fluid. According to its magnitude, tissue pressure offers resistance to capillary filtration and accumulation of an excessive amount of extravascular fluid, and promotes capillary absorption and lymph flow. Tissue pressure is proportional to the density and elasticity of the tissue, increases with the rapidity of capillary filtration, and becomes less as the elasticity of the tissue is lost by long continued distention of its interstitial spaces by edema fluid.[1] An increase in tissue pressure is resisted by the fascial planes, integument covering the part, and bony enclosures of the body. Tissue pressure may be increased by application of physical pressure to the surface of the part. Other pressure variables which affect the origin, distribution and absorption of extravascular fluid are arterial and venous capillary blood pressure, osmotic pressure, hydrostatic pressure and atmospheric pressure.

Anatomically the lymph pump mechanism may be divided into two parts: one being the lymph bed of the nose and paranasal sinuses, and the other the retropharyngeal lymph nodes into which the lymph bed drains. Each of these parts when involved in pathologic states manifest their functional pathology by clinical signs and symptoms which are of diagnostic value.

The retropharyngeal nodes are from two to 10 in number during childhood. These are divided into medial and lateral groups. The medial group atrophies during adulthood, usually leaving only one of the lateral retropharyngeal nodes on one side. Through these nodes drains all of the lymph from the structures developed from the first branchial pouch, *i.e.*, the middle ear, Eustachian tube, adenoid, nasopharynx, nasal cavities and paranasal sinuses. Lymph from these structures passes through the lymph capillaries and pathways in the mucosa of the nose and nasopharynx into either pretubal lymphatic plexus. Efferent vessels from these plexuses pierce the pharyngeal aponeurosis and enter the retropharyngeal nodes. The pretubal lymphatic plexuses anastomose freely through channels which traverse the pharyngeal surface of the soft palate. This free communication between the pretubal plexuses is important to an understanding of the bilateral character of lymph edema occurring during acute blockage of the retropharyngeal nodes and the retrograde metastasis of cancer cells in this region; also, these channels may be the route of flow from one nasal cavity to the other of the increased amount of extravascular fluid present in the tissues during certain pathologic states.[3]

The physic qualities and anatomic structure of the sinal mucosa and turbinal tissue are specifically designed and adapted for the purpose of promoting lymph flow from the stroma of the mucosa and drainage of sinal secretion. To understand better this adaptation, it is important to recall that all air-containing cavities of the body, when they become severely infected, tend to lose their air by becoming filled with edematous tissue and/or secretion. As air is driven from a cavity the tissue pressure of the tissues in the cavity is raised and pulsations from the arteries and dilated arteriovenous anatomoses are transmitted to the lymph vessels through an incompressible fluid medium and thus lymph is pumped toward the lymph nodes. The perfection of this process usually is related to or varies with the virulence and invasive characteristics of the offending agent.

To facilitate discussion of the adaptation of the lymph bed to the function of promoting lymph flow, it is useful to divide it into two functionally related parts; one being the sinal mucosa and the other the turbinal tissue. The normal sinal mucosa is "seldom more than 0.02 mm. in thickness"[4] and consists of a very thin stroma of closely placed fibroblasts surmounted by the pseudostratified ciliated epithelium. On section the cells of the stroma appear to be placed so close to each other that there are relatively few intercellular or interstitial spaces present. When the stroma of the normal mucosa is teased apart, the cells separate into layers with the lines of cleavage appearing parallel to the epithelial surface. This sparse cellular and tightly layered construction of the stroma of the normal sinal mucosa accounts for the facility with which the mucosa becomes edematous, approximates the capacity of the sinus and thereby raises its tissue pressure. Also, the scarcity of interstitial spaces and blood vessels in the normal sinal mucosa precludes absorption from its surface. Some form of conditioning process which dilates its interstitial spaces and provides more blood supply is necessary before absorption can occur through this membrane. Conditioning of this membrane for absorption will be considered under allergic sinusitis.

Drainage from the normal sinus is accomplished chiefly by ciliary action. During pathologic states drainage is accelerated by reduction in the size of the sinal cavity by swelling of the sinal mucosa. During inflammation the degree of swelling of the sinal mucosa is greater in that part of the mucosa which is far removed from the ostium of the sinus and swelling of the mucosa is least in the region of the ostium. This provides for drainage of the sinal cavity by the edematous mucosa forcing the secretion to or through the ostium of the sinus. When swelling of the mucosa becomes extreme, portions of the mucosa may prolapse through the ostium as polypoid protrusions.

It is important to an understanding of this factor in sinal drainage to consider the relation of the anatomic structure of the sinal mucosa to its tissue pressure. The opinions expressed here have been derived chiefly from studies of the mucosa of the maxillary sinus. Sections of the mucosa from various parts of this sinus show it to contain more glands, blood vessels and lymph vessels near the ostium. Also, it is thicker and more dense in the area of the ostium than it is in areas far removed from the ostium. Since tissue pressure resists capillary filtration and is related to the density and elasticity of tissue, it is evident that following a given uniform stimulus to increased capillary filtration, the filtrate appears first and dilates to a greater degree the interstitial spaces in that portion of the sinal mucosa which are away from the ostium of the sinus. As the edema of the sinal mucosa progresses, the mucosa displaces the air from the sinal cavity and forces the secretion toward the region of the ostium of the sinus. During this process the ostium remains patent, due to the relatively greater tissue pressure of its mucosa.

The variables which affect capillary filtration are the colloidal osmotic pressure of the blood, the permeability and blood pressure of the arterial and venous capillaries and the tissue pressure. If other factors remain constant and there is lowering of the colloidal osmotic pressure of the blood as in simple hypoproteinemia, increased capillary filtration occurs first into areas of the body which have the least tissue pressure. This is commonly seen clinically in the edema in the loose tissue around the eyes during hypoproteinemia accompanying albuminuria, and in the feet and ankles where hydrostatic pressure and gravity play a part in increasing capillary filtration and collection of fluid in the interstitial spaces. Gravity also may play a part in the distribution of edema fluid in the sinal mucosa. If the sinal mucosa is very edematous the fluid may become relatively free, *i.e.*, not a part of the tissue gel. As tissue fluid becomes free to move in the interstitial spaces of the sinal mucosa it gravitates to the most dependent portion of the sinus. Shifting by gravity of this edema fluid to the most dependent part of the sinus also is a factor in bringing secretion to the level of the region of the ostium and facilitates sinal drainage. This clotting tissue fluid often may be aspirated from the sinal mucosa through a needle but should be differentiated from similar appearing nonclotting cystic fluid or the fluid portion of the secretion contained in the sinal cavity.

In cadavers with generalized edema and otherwise normal sinuses which have undergone rigor mortis in dorsal decubitus, the mucosa of the posterior walls of the sinuses is much more edematous than the mucosa on the other walls. There is some evidence that the tissue pressure of the sinal mucosa is probably less than that of any other tissue of the body. If this be true during severe hypoproteinemia the sinal mucosa is the first tissue to become edematous and the last tissue from which the edema fluid is absorbed. During X-ray examination using contrast media, this type of edema has been interpreted erroneously as primary sinal pathology.

The intranasal structures aid in changing the extravascular fluid and promoting lymph flow by the same fundamental methods utilized by the sinal mucosa. These structures differ from the sinal mucosa anatomically and physiologically in that the nasal mucosa has greater normal tissue density and higher normal tissue pressure, while the turbinal tissue in addition to these qualities possesses inherent erectility and elasticity and its smooth muscle fibres provide for more enduring contractility.

All of the lymph from the mucosa of the sinuses must pass through the lymph channels in the nose en route to the retropharyngeal nodes. In the normal nose the tissue pressure of the turbinates and nasal mucosa is so balanced against capillary filtration that only enough fluid and protein is filtered into the tissues to meet the physiologic requirements; therefore, the intranasal tissue is able to remove the nomal extravascular fluid without abnormal turgescence. Following irritation or infection of the nasal mucosa, there is an increase in the amount of interstitial fluid to be removed from the lymph bed, so the intranasal structures drive air from the nasal cavity by swelling against their surrounding bony walls. The lymph can then be pumped from the lymph bed by pulsations transmitted from the arteries to the lymph vessels through an incompressible fluid medium.

The anatomy and physiology of the normal nose provide for nasal cavities either of which alone can transmit sufficient air to the lungs for normal, quiet respiration while the opposite cavity is closed. During moderate irritation or infection, swelling of the turbinal tissue alternates from one nasal cavity to the other, *i.e.*, the right nasal cavity will close while respiration occurs through the left, then the left will close and respiration occurs through the right. This alternating cycle of opening and closing of the nasal cavities was described by the Hindus during the Vedic Period (1500 B.C.). A British anatomist, Hemchandra Sen, in 1901, described how one and then the other of his own nasal cavities would alternately open and close every few hours. His observations were made by sticking his tongue up behind his soft palate and examining the condition of his turbinates, noting that one side of his nose would close, alternating with the other side.[5]

The alternate opening and closing of the nasal cavities may be interrupted by gravity. In lateral decubitus, the nasal cavity which is inferior will close, while in dorsal decubitus both nasal cavities will remain open when there is only a moderate amount of extravascular fluid to be removed from the lymph bed. When gravity fails to affect or aggravates intranasal tissue turgescence, usually the reaction to the agent which caused the intranasal swelling has been severe. Also, gravity may have little effect during partial or complete lymphatic blockage of the retropharyngeal lymph nodes, or when the protein content and colloidal osmotic pressure of the edema fluid in the sinal mucosa is very high.

The presence, location, cytology and physical characteristics of nasal and sinal secretion, the color and swelling of the sinal mucosa, and the anatomic construction of the nasal cavities are diagnostic criteria important during rhinitis and sinusitis; however, the degree and location of swelling or turgescence of the turbinal tissue also is a most valuable diagnostic aid in nasal and sinal disease.

The functional pathology of the turbinates consists of their becoming turgescent and increasing their tissue pressure as an auxiliary or aid to the sinal mucosa as the mucosa for functional purposes needs to increase its tissue pressure.

The character and degree of turgescence of the turbinates are related to the type and extent of lymphatic blockage present in the sinal mucosa. Also, the tissue pressure of the sinal mucosa, the colloidal osmotic pressure of the secretion and interstitial fluid of the sinal mucosa, and the presence in the tissue spaces of the sinal mucosa of relatively unremovable substances are important factors affecting turgescence of the turbinates. During purulent infections these relatively unremovable substances, until more information concerning their true nature is available, may be regarded as plasma colloids and bacterial products, while in allergic states they may be regarded as air- or blood-borne protein antigens. If the allergic state is long continued, bacterial antigens from secondary infection also may be present. During acute purulent states the sinal mucosa becomes edematous and increases its tissue pressure in direct proportion to the colloidal osmotic pressure of its edema fluid. If the colloidal osmotic pressure of the secretion in the sinus is high and holds in the stroma of the mucosa fluid of high protein content the turbinates will remain turgescent until the secretion is drained from the sinus and the colloidal osmotic pressure of the edema fluid in the sinal mucosa is reduced. As the protein in the edema fluid is carried away, there is no longer need for an accessory swelling of the turbinates to increase the tissue pressure as an aid to removal of components of the extravascular fluid. To understand the interrelationship of these variables it is important to consider their relation to lymph flow.

Lymphatic blockage in the mucosa of the lymph bed is the principal factor in determining the relation of turgescence of the turbinates to pathology in the sinuses. It may be recognized in three stages: acute, subacute and chronic. Hindrance to lymph flow in either of these stages may be partial or complete. Acute lymphatic blockage occurs as fibrin, cellular detritus, and round cells infiltrate the intercellular and interstitial spaces of the sinal mucosa. During this stage blockage of the interstitial spaces is usually so complete that nothing except certain diffusable toxic products pass through the walled off area. This results in retention in the mucosa of a highly proteinized tissue fluid rich in albumin with correspondingly high colloidal osmotic pressure. During this stage the turbinal tissue of the same side is constantly and completely turgescent, or as alternating nasal stenosis ensues the turbinates on one or both sides may recede incompletely during the cycle of alternation.

During the subacute stage of lymphatic blockage the fibrin and cellular detritus have been absorbed, leaving only the round cell infiltration. In this stage of lymphatic blockage the interstitial spaces in the stroma of the sinal mucosa are so filled with round cells that there is little room for excess extravascular fluid. The turbinates during this stage may appear normal or show alternating turgescence during acute exacerbation of infection. Subacute lymphatic blockage may continue for years without seriously interfering with nutrition of the mucosa. This type of mucosa may be returned to a semblance of normal by application of physical pressure to its surface.[6] During chronic lymphatic blockage in the sinal mucosa, fibrosis of the stroma of the mucosa is present and there are relatively few interstitial spaces to hold fluid. This type of blockage usually terminates in loss of nutrition to the glands and ciliated epithelium of the mucosa. Often this type of mucosa is seen surrounding a septic cavity. The turbinal tissue in this stage is not turgescent because there is little extravascular fluid to be removed from the stroma of the sinal mucosa. Also, the pathways of exit for lymph from the sinal mucosa are so blocked by fibrous tissue that there is no stimulation to the turbinates to increase their tissue pressure by becoming turgescent.

The clinical application of the foregoing principles of nasal physiology and functional pathology will now be applied to certain types of nasal stenosis due to turgescence of the turbinates. Turgescence of the turbinates will be classified as alternating, when it changes from one nasal cavity to the other, and as intermittent, constant or complete when either or both nasal cavities are closed intermittently, constantly or completely.

I. *Excessive Intranasal Medication.* Patients suffering from this frequently self-administered condition usually begin with mild bacterial or allergic rhinitis and alternating nasal stenosis. They usually give a history of having administered "nose drops" or inhalations at increasingly frequent intervals until they have bilateral constant, complete nasal stenosis. These patients have overstimulated the contractile power of their turbinal tissue, and must stop their medication and necessarily go through a period of constant nasal stenosis until their turbinates regain their contractility. In this condition it is important to impress on the patient that during certain pathologic states it is necessary for the nasal cavities to become blocked as part of their functional pathology before they can return to normal; therefore, if they continue their intranasal medication they only postpone the time when the blockage will occur. Some of these patients are aided greatly by irrigation of their maxillary sinuses and evacuation of trapped secretion.

II. *Nasal diphtheria and glanders* produce acute lymphatic blockage of the retropharyngeal lymph nodes and cause constant complete nasal stenosis due to lymph edema of the lymph bed. Frequently this type of swelling of the intranasal tissues cannot be reduced with adrenalin, cocaine or other commonly used shrinking agents.

III. *Acute purulent sinusitis* is recognizable clinically in three stages:

1. *Empyema of the Sinus.* In this stage the sinus contains secretion having a protein content frequently as high or higher than the protein of the blood plasma with a corresponding high colloidal osmotic pressure. This high osmotic pressure secretion holds in the stroma of the sinal mucosa interstitial fluid high in protein. This is reflected intranasally in marked turgescence of the turbinates on the affected side as the lymph pump mechanism attempts to remove the highly proteinized tissue fluid. The turgescence of the turbinates in this stage is due to marked dilatation of their cavernous bodies, lymph edema of the intranasal mucosa and blood vessel engorgement. As lymphatic blockage in the sinal mucosa becomes complete the turgescence of the turbinates may subside slightly.

If the sinusitis is unilateral the nasal stenosis then may become unilaterally intermittent, or if it is bilateral it may alternate from one nasal cavity to the other as the process passes into the draining stage.

2. *Draining Stage.* In this stage the colloidal osmotic pressure of the sinal secretion has fallen below that of the edema fluid in the sinal mucosa. This allows the colloids to be removed from the edema fluid in the sinal mucosa by the lymphatic system and the water and electrolytes to be absorbed by the veins. During this stage, if the sinusitis is unilateral the nasal stenosis due to turgescence of the turbinates is intermittent on the affected side. If the sinusitis is

bilateral, alternating nasal stenosis occurs, excepting when the turbinates are affected by gravity. The effect of gravity in this stage is evidenced clinically by placing the patient with normally constructed nasal cavities in lateral decubitus and noting that the nasal cavity uppermost is open while the inferior cavity is closed by turgescence of the turbinates. Also, if the patient is placed in dorsal decubitus, both nasal cavities will open unless there is partial or complete lymphatic blockage of the retropharyngeal lymph nodes. There is no exit from the nose anteriorly for lymph or excess extravascular fluid; therefore, if the patient is placed in ventral decubitus, both nasal cavities may close if the sinusitis is bilateral. Factors such as toxins, tissue destruction and specific invasive characteristics of certain organisms may alter these clinical findings.

3. Resolution Stage. The sinal secretion has almost disappeared in this stage and has become definitely separately into its clumped and fluid portions. The osmotic pressure of the secretion is so low it is negligible, and the sinal mucosa has become sufficiently edematous to increase its tissue pressure to a magnitude which promotes lymph flow. Often the mucosa is so edematous it completely fills the sinal cavity. When this occurs in the maxillary sinus and the sinus is punctured with a needle under the inferior turbinate, the needle may enter the edematous mucosa and as irrigation is attempted part of the edematous mucosa may be forced against the ostium and block the irrigation. Clinically it is important to understand that this marked swelling of the sinal mucosa is an essential part of its functional pathology. In the stage of resolution, even though the sinal mucosa is very edematous, if the mucosa is not mistreated it will rapidly return to normal. The alternating or intermittent nasal stenosis which ushers in the stage of resolution gradually subsides until the turbinates return to normal.

IV. Chronic Purulent Sinusitis. The protein content and osmotic pressure of the sinal secretion during chronic purulent sinusitis are high, but the interstitial tissue spaces of the sinal mucosa have become filled first with round cells and later with fibroblasts which have blocked the lymph pathways. This type of mucosa contains little protein that can be removed by the lymph pump mechanism; therefore, there is no increased turgescence of the turbinates or nasal stenosis to betray the presence of purulent secretion in the sinus.

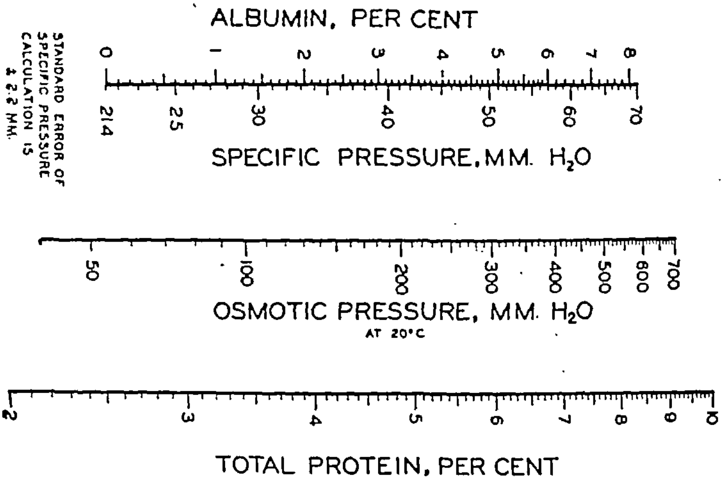

Fig. 1. "Nomogram. A straight line drawn through the proper points on the albumin and total protein scales will intersect the middle scale at the value of the total osmotic pressure calculated from the formula, $P = C(21.4 + 5.9A)$. Where P is the osmotic pressure in millimeters of water, C is the total protein concentration and A is the albumin concentration in grams per 100 cc. The standard error of calculation is ± 5 per cent." (From H. S. Wells, J. B. Youmans and D. G. Miller, Jr., Jour. Clin. Invest., 12:1103, Nov., 1933.)

V. Allergic Rhinitis and Sinusitis. Purulent rhinitis and sinusitis is primarily a disease of the surface of the mucosa, while allergic rhinitis and sinusitis primarily affects the stroma of the mucosa. These two conditions also differ in their stimulation to increased permeability of the capillaries. During purulent disease the increased permeability of the capillaries allows a relatively large quantity of protein to filter into the tissues, while during allergic disease, although the edema may occur very suddenly and extensively, the protein content of the capillary filtrate is relatively low. Clinically, this is evidenced by the rapidity of absorption, probably principally by the veins, of edema occurring during certain allergic states such as urticaria and angioneurotic edema, while the highly proteinized filtrate occurring during purulent disease requires more time for processing and removal of the protein through the lymphatic system. One outstanding difference between purulent and allergic disease of the sinuses is to be found in the relative absence of lymphatic blockage in the lymph bed during pure allergic sinusitis when compared to that present during *purulent sinusitis*. The turbinates become turgescent to aid the sinal mucosa in increasing its tissue pressure; however, the turbinates respond in this manner only if the interstitial tissue spaces in the sinal mucosa between the turbinates and the sinus are "open." During allergic states when relatively unremovable antigenic substances are present in the sinal mucosa the turbinates may manifest increased turgescence constantly until the antigen is removed or until lymphatic blockage occurs in the interstitial spaces of the stroma of the mucosa. Apparently in certain instances the tissue must be conditioned before allergic manifestations can occur in certain areas. Condition-

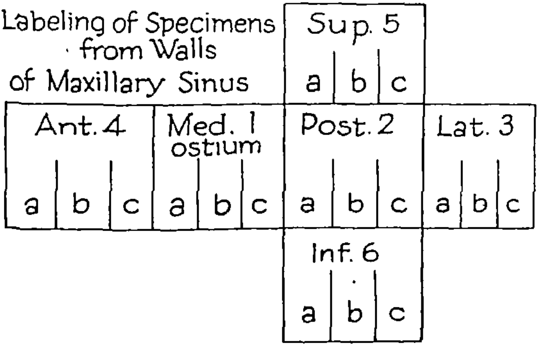

Fig. 2. Method of labeling specimens of mucosa procured from the maxillary sinus for study of normal and pathologic states.

ing may result from frequent application of an antigen to the same area. Any agent which opens the interstitial spaces of the sinal mucosa to absorption and retention of antigens may act as a conditioning factor. Repeated episodes of purulent or allergic rhinitis may condition the tissue for the occurrence of allergic sinusitis. This is in accord with the Arthus phenomenon which is one of the bases of allergic reactions. "Arthus found that if he injected antigen into animals subcutaneously at definite intervals the first few injections were absorbed readily but later injections were not absorbed. Instead, they became surrounded by an area of inflammation and often tissue necrosis occurred in the areas of recent injection."[7]

Following the reaction of the nasal and sinal mucosa in the manner of the Arthus phenomenon, or one of its variations, the fate of antigenic substances which are brought in contact with its surface or brought by the blood stream to its interstitial spaces is similar to homologous antigens injected into the skin, *i.e.*, one fraction is absorbed into the blood stream either rapidly or slowly and another fraction remains in the area of injection and becomes attached to the cells of the body until it becomes proteolyzed, bacteriolyzed or extruded.[8] Absorption from the sinal mucosa of these antigenic substances of products of their ageing process may produce bronchial asthma in accord with the smooth muscle reaction of Schultz and Dale, or they may produce immunologic shock in accord with the Schwartzman phenomenon. This is in accord with the observations of Landsteiner that "simple chemical substances probably form conjugates with the animal's own proteins, forming new antigens. The presence of these new antigens resulted in sensitization."[7]

1. Acute allergic rhinitis due to inhaled or blood-borne antigens presents alternating nasal stenosis which varies in its severity with the amount of antigen encountered and the sesitivity of the patient. If the allergic reaction is severe or if the patient has indulged in excessive intranasal medication, complete constant nasal stenosis may ensue. Constant complete nasal stenosis occurring during or immediately following the hay fever season often means that the patient has developed acute allergic sinusitis.

2. Acute allergic sinusitis presents constant complete nasal stenosis as the turbinates become turgescent to aid in removing relatively unremovable antigen from the interstitial spaces of the sinal mucosa. Irrigation of the maxillary sinuses in these patients evacuates a small amount of white mucoid secretion containing a marked concentration of eosinophiles. In certain instances particulate matter may be present in the sinal secretion. Irrigation of the maxillary sinuses, preferably by antrum puncture under the inferior turbinate, is of great value in promoting resolution of this type of sinusitis. The clinical improvement of these patients has been much more rapid following irrigation of the maxillary sinus by puncture instead of irrigation through the ostium. This may be due in part to draining of tissue fluid from the fistula made in the edematous sinal mucosa during puncture of the sinus.

The first attack of acute allergic sinusitis is one of the most important episodes in the life of the allergic patient. Often the patient has bronchial asthma for the first time with this type of sinusitis. If the sinus secretion is evacuated promptly after the asthma begins, the asthma usually subsides rapidly; however, if the secretion is allowed to remain in the sinuses the asthma may become protracted and the sinusitis may lapse into the subacute stage.

3. Subacute allergic sinusitis presents a variety of types of nasal stenosis due to turgescence of the turbinates. One complaint most of these patients have in common is alternating or constant nasal stenosis which often is accompanied by symptoms which the patient interprets as "catching cold." The mucosa of the maxillary sinus in this stage may be uniformly edematous and less than 1 mm. in thickness. Apparently the sinal mucosa contains substances which do not stimulate it to increase its tissue pressure sufficiently to promote their removal. In this type of sinusitis the turbinates may show increased turgescence constantly as they attempt to aid the sinal mucosa to increase its tissue pressure.

The subacute stage of allergic sinusitis may last for years and often is accompanied by bronchial asthma which may occur episodically at first and then may become constant. Apparently the sinal mucosa in this stage contains antigen which is undergoing proteolysis, the very slight edema present being a necessary part of this process. X-ray studies of the sinuses, using contrast media, often fail to reveal this slightly edematous mucosa but it can be visualized with an antroscope. This type of sinal mucosa may be returned to a semblance of normal by application of local pressure to its surface.

Frequently the only signs or symptoms presented by the patient during subacute allergic sinusitis are those related to increased turgescence of the turbinates, with or without nasal eosinophilia. Since there is no excess secretion present in the sinuses and the very slight uniform edema of the sinal mucosa is not demonstrable by X-ray, it is easy to overlook the relationship of turgescence of the turbinates to the tissue pressure of the sinal mucosa and erroneously classify many of the early cases of subacute allergic sinusitis as rhinitis due to vasomotor, neurogenic, toxic, endocrine, etc., etiology.

The slight persistent edema of the sinal mucosa present during subacute allergic sinusitis is the first phase of the constantly progressive pathologic changes which are characteristic of allergic disease of the sinuses. The pathologic changes in the mucosa which follow this phase are related to the frequency and severity of secondary infection. Increased turgescence of the turbinates accompanies each episode of secondary infection of the edematous sinal mucosa. Following repeated infection, partial blocking of the interstitial spaces of the stroma of the mucosa by round cells and fibroblasts occurs and stagnation of the edema fluid in the mucosa ensues. This type of mucosa may become so edematous that it may fill entirely the sinal cavity. These pathologic changes usher in the chronic phase of allergic sinusitis.

4. Chronic allergic sinusitis differs pathologically from chronic purulent sinusitis in that the fibrosis which occurs in the stroma of the sinal mucosa during purulent sinusitis is more uniform and complete, while the fibrosis which occurs

in the stroma during chronic allergic sinusitis is irregular and incomplete. This results in the formation of alternating areas of fibrosis and dilated tissue spaces interspersed throughout the stroma of the mucosa. As infection occurs in this type of mucosa and the tissue pressure rises, polypi protrude from the areas of dilated tissue spaces. During this stage the turbinates remain turgescent until lymphatic blockage in the interstitial spaces of the stroma of the sinal mucosa occurs. If lymphatic blockage in the sinal mucosa is incomplete and the sinal cavity is totally or partially filled with polypi, the turbinates may show constant increased turgescence as an aid to removal of protein or bacterial products or antigens from the tissue fluid of the sinal mucosa. Long continued swelling of the turbinates may result in loss of their contractility so that they become boggy and unresponsive to the usual stimuli. As polypi fill the nasal cavities and increase the intranasal tissue pressure the turbinates may become compressed and regain some of their contractility.

SUMMARY.

1. The lymph pump mechanism of the nose and paranasal sinuses essentially consists of the anatomic and physiologic adaptation of the sinal mucosa and turbinal tissue to the *process of adjusting* their tissue pressure to a magnitude which promotes the flow of lymph from their tissue spaces. Interrelated variables which enter into this mechanism are:

1. The relation of the normal and pathologic tissue pressure of the turbinal tissue to the tissue pressure of the sinal mucosa.

2. The relation of the colloidal osmotic pressure of the sinal secretion to the colloidal osmotic pressure of the interstitial fluid in the sinal mucosa.

3. Lymphatic blockage in the sinal mucosa or in the retropharyngeal lymph nodes.

4. The presence of relatively unremovable protein or bacterial products or antigens in the turbinal tissue or sinal mucosa. This factor is important during both purulent and allergic states.

An understanding of the lymph pump mechanism is useful clinically as an aid in interpreting intranasal findings as they reflect sinal pathology.

The turbinal tissue and sinal mucosa increase their tissue pressure to a magnitude which promotes lymph flow by swelling against their inherent elasticity and contractility and their bony enclosure.

Normally, lymph flows in most of the body by active and passive motion, muscle contraction, massage, gravity, tissue pressure and pulsations transmitted from the arteries to the lymph vessels.

The nose and paranasal sinuses are deprived of active and passive motion and massage to promote lymph flow. Instead, they depend upon tissue pressure, gravity and transmitted arterial pulsations. The latter is important during pathologic states as the tissues swell and obliterate the air-containing cavities so that arterial pulsations can be transmitted to the lymph vessels through an incompressible fluid medium.

The turbinal tissue becomes turgescent and increases its tissue pressure as an auxiliary or aid to the sinal mucosa as it becomes necessary for the sinal mucosa to increase its tissue pressure in an effort to remove substances from the interstitial spaces of the stroma of the sinal mucosa.

Reaction of the turbinates in this manner is governed principally by the freedom with which fluid may move through the interstitial spaces of the nasal and sinal mucosa. During complete blockage of the interstitial spaces in the sinal mucosa to the flow of lymph and extravascular fluid, there is no stimulus to the turbinates to become turgescent. If the interstitial spaces in the sinal mucosa are open and contain relatively unremovable substances the turbinal tissue will remain turgescent until the sinal mucosa has increased its tissue pressure to a magnitude which effects their removal or until these substances become proteolyzed or bacteriolyzed and removed.

Correlation of clinical findings during certain pathologic states with certain principles of physiology and functional pathology of the turbinates and sinuses has been attempted.

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UNIVERSITY OF ILLINOIS POSTGRADUATE COURSE.

The University of Illinois College of Medicine, Department of Otolaryngology, announces its annual postgraduate course in Basic Otolaryngology beginning Oct. 2, 1950. A full time program of instruction is followed for those intending to prepare for special practice. The course terminates on June 15, 1951. For information and application blanks, contact the University of Illinois College of Medicine, Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

THE USE OF A WETTING AGENT IN LARYNGOTRACHEOBRONCHITIS WITH REPORT OF CASES TO DATE.*

JAMES R. GORMAN, M.D.,
Lynchburg, Va.

We are all faced repeatedly with the terrible problem of laryngotracheobronchitis. Before and after tracheotomy a great many of these cases present severe dryness of the trachea and bronchi, with or without crusting *below* the tracheotomy tube and sometimes with a thick, tenacious secretion which cannot be coughed up by the child.

This past Fall we have had in our area an epidemic of severe proportions, totaling among the pediatricians nearly a hundred cases, three of which required tracheotomies as lifesaving measures. Two of them occurred on the same day and the third within a 10-day period. My report has to do primarily with this third case.

CASE REPORT.

Mickey D., white male, 28 months old, was admitted to Virginia Baptist Hospital Sunday, Nov. 6, in a critical condition, requiring immediate tracheotomy; temperature, 102°. Following tracheotomy, the child did fairly well, but the trachea was perfectly dry with very little secretion coming through the tube on coughing, even with the use of normal saline dropped into trachea and suctioned back. Seen at 8:30 A. M., Tuesday, the child was in good condition as to color, breathing and other signs, except dry, severe cough. At 4 P. M. this same day I was called hurriedly to the hospital and found the child in dying condition. The tube was clear, but the child was cyanotic and gasping for air. I thought he would die before we could get him to the operating room, where we had the proper materials, light, suction, etc.

I would like to emphasize one important little point in connection with these dry cases, especially with thick, tenacious discharge and crusts, and that is that I much prefer to use the malleable copper Jackson suction tube in preference to the bronchoscope. I used this tube with suction attached, introduced through the tracheotomy wound, and recovered some very thick, tenacious material which the child was unable to cough

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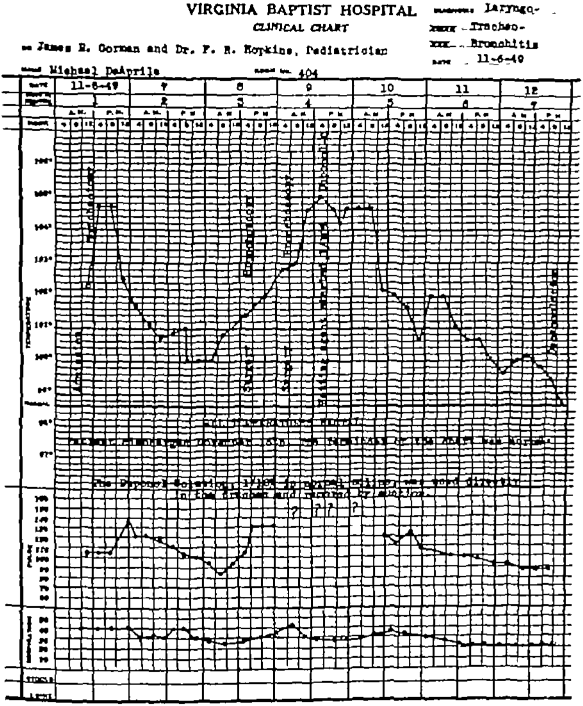
*Read at the combined meeting of the Middle and Southern Sections, American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn. Jan. 17, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 23, 1950.

up. Repeating this procedure several times, considerable material was removed, which, when placed on a towel, hardened in about 10 minutes to the consistency of hardened glue. After its removal the tracheotomy tube was reintroduced and the child watched for half an hour and then returned to his room. His temperature had increased from 100 to 102.° I was unable to have slides made but the original hospital chart is shown below.



At 3 A. M. the morning of Wednesday I was called back to the hospital, and the above procedure had to be repeated as a lifesaving measure. This was the morning of the fourth hospital day. The child was receiving large doses of penicillin and highly humidified air in croup tent, and aureomycin.

We did not have a resident at this hospital. My office is five miles and my home seven and one-half miles from the hospital. Fearing the child would die before I could reach him, I called Dr. Fletcher D. Woodward at the University of Virginia and told him my problem. I wanted to send the child there as he has trained residents, but he thought we were doing everything possible. In the discussion with Dr. Woodward, he asked whether I was using saline in the trachea. This, of course, had been done, but it had no effect in this particular case. I asked him if he had ever used a wetting agent in the saline. He said "he had not but thought it would be a good idea." The problem then was to obtain the proper wetting agent. I consulted the U. S. Pharmacopea and found that Duponol-C was listed and approved by the Pure Food and Drugs Commissioner. I am indebted to Mr. N. T. Gorchoff, of the Virginia Specialty Co., for furnishing me with the original supply of Duponol-C which was used in normal saline one-tenth per cent and instilled directly into the trachea through the tracheotomy tube, using a medicine dropper, followed by immediate use of suction, withdrawing fluid mixed with secretions. From the time the solution was started the child began to improve rapidly and was able to cough up the material which we previously had to remove on two occasions. The one-tenth per cent solution of Duponol-C was started at 6 P. M. on Wednesday; on Saturday I was able to decannulate the child, and on Wednesday of the following week was able to discharge the patient.

The cases which present these dry crusts and crusting material in the trachea and bronchi offer a big problem. Cases have died with the bronchoscope in place and in addition to this danger the scope may push the crusts down into the trachea and the main stem bronchi, and the child may die on the table. Dr. Woodward told me he had had one die on the table, being unable to remove the crusts. The small calibre Jackson malleable copper suction tube is a perfectly safe instrument to use through the tracheotomy wound if gently introduced and removed when pressure gauge shows resistance and the secretion or crust removed from the end of tube.

This particular patient had a 10-day hospital stay. The X-rays of chest were essentially negative. The case was treated in consultation with Dr. F. Read Hopkins, pediatrician.

About two weeks later I called Dr. Woodward to report to him the progress made in this case. He said, "I have just left the hospital and have seen a child with laryngotracheobronchitis who needed immediate tracheotomy, and have told the resident to do it, but I will call the hospital and tell them to get some Duponol-C and put it in the water being vaporized." A letter from him a few days later says, "The child admitted with laryngotracheobronchitis and who apparently needed an immediate tracheotomy, we introduced the wetting agent in water or saline of the vaporizer, at the end of three hours the child was much improved and in five hours was out of danger."

After these two experiences we decided to try its use further with such cases and I wish you would do the same.

Not including the child on whom a tracheotomy had been done, but including Dr. Woodward's case, we now have seven children on whom the drug has been used in the water being vaporized with equally dramatic results and on whom it was not necessary to do tracheotomies. Two cases have been added to the five reported at Memphis.

Becoming interested and knowing a little about wetting agents and the many uses to which they are being put was the reason for my thinking of it in this connection. Proper agents of this type are being used in eye solutions to lower the surface tension and keep the surface moist for a longer period of time.

The fire departments of many cities are using the agents, which in proper dilution are carried in the 250 gallon tanks. This amount of water properly treated will do approximately what 5,000 gallons of water would do.

I wrote to the American Cyanamid Co. regarding wetting agents in common. They promptly sent me a reprint of the work they had done on animals at their Stanford Research Laboratories. They produce the Aerosols O. T. The reprint is rather long, but in the tests they used rabbits, dogs, monkeys and rats. The animals were fed the wetting agent for a period of six months. They state "that although a total of 221 histological preparations of tissue were made from nine rats, six dogs, three monkeys and four rabbits on high dosage were studied, no significant pathology was observed. This is consistent with the observations of Lorenz, Schenkin and Stewart in mice which received the Aerosol O. T. by mouth."

I was also sent their brochure on wetting agents which gives a great deal of information about them and their uses generally. Aerosol O. T. is Dioctyl Sodium Sulfosuceinate. Duponol-C is Sodium Lauryl Sulphate ($CH_2(CH_2)_{10}CH_2OSO_3$ Na) U. S. P. This particular agent or similar ones are being used in shampoo mixtures, tooth pastes, cosmetics, liquid dentifrices and other materials used about the skin.

All of the cases on which the drug was used were not mine, but the agent was used under my direction.

The two and one-half-year-old son of one of our eye, ear, nose and throat men was seen Dec. 24 about 3 P. M. and it looked very much as though a tracheotomy would be needed. The child was in a very small cubical-like room, closed off with heavy drapery. Humidity at 3 P. M. was 65, using an open bucket on a hot plate. At 3:30 the Duponol-C was added, about one-fifth of a teaspoon to the gallon, and in 30 minutes the humidity had increased to 90. By 6:30 P. M. the child was greatly improved and apparently out of danger. Seen Christmas Day, he was running around in his bed, playing with his Christmas toys, and made an uneventful recovery. The room was so moist that the wallpaper had loosened and the doctor told me he would have to redecorate the room.

The important thing is that the moisture is essential and should be maintained. The use of a wetting agent (in these cases Duponol-C) seems to greatly increase the moistening effect, both of the saline one-tenth per cent used directly into the trachea, when the tracheotomy has been done, and in the water being vaporized, whether by steam or actual vaporization. For the comfort of the patient and attendants, the temperature of the room should be controlled, especially where steam is being used.. There should be some ventilation of the room.

Several of our drug stores now stock Duponol-C. Whether this is the best agent to be used remains to be seen and I hope that you gentlemen who have at your disposal laboratory and clinical facilities will use this suggestion and make further reports on studies of its usefulness. Anything which is helpful in laryngotracheobronchitis will be worthwhile.

In talking with Dr. Woodward over the telephone, Feb. 17, he said they had had several cases in which the drug had been used by vaporization and they had been able to avoid tracheotomy on all of them and that there would be subsequent reports on the use of the drug.

1108 Allied Arts Building.

In Memoriam

FRANCIS R. PACKARD, M.D.,

1870-1950.

Dr. Francis R. Packard, prominent medical librarian and pioneer otolaryngologist, died April 18 at Pennsylvania Hospital at the age of 80.

Born in Philadelphia, the son of Dr. John Hooker and Elizabeth Wood Packard, he graduated from the University of Pennsylvania in 1889 and three years later received his medical degree there.

Dr. Packard was first assistant surgeon of the Second Pennsylvania Volunteers during the Spanish-American War, and in World War I was center consultant for the district of Paris.

In 1930, Dr. Packard was elected president of the American Laryngological Association; in 1931, he was president of the College of Physicians of Philadelphia, and in 1936 was president of the American Otological Society. He was also a former president of the Musical Fund Society of Philadelphia and associated with various library groups. He was a member of the Academy of Stomatology of Philadelphia, a member of the Philosophical Society, University Barge Club, Franklin Inn, Philadelphia Shakespeare Society, Wistar Association and the Historical Society of Pennsylvania.

He was the proud owner of one of the largest medical libraries in this country and the author of many formidable papers in his specialty, but his outstanding contribution to otolaryngologic literature was perhaps his History of Medicine in the United States. During the early years of THE LARYNGOSCOPE, Dr. Packard was quite active on its editorial staff and was a frequent contributor to its pages.

As honorary keeper of the archives of Pennsylvania Hospital, he had at his command valuable data from which he compiled a history of the hospital from 1751 until 1918.

He is survived by four daughters: Mrs. E. Perot Bissel, Jr., of Haverford; Miss Elizabeth Packard, of Philadelphia; Mrs. Peyton R. Biddle, of Rosemont, and Mrs. John H. Rhein, of Seaford, Del.

BOOK REVIEWS.

Fundamentals of Otolaryngology. A Textbook of Ear, Nose and Throat Diseases. By Lawrence R. Boies, M.D., Clinical Professor of Otolaryngology, Director, Division of Otolaryngology, University of Minnesota, and associates Charles E. Connor, M.D., Anderson C. Hilding, M.D., Jerome A. Hilger, M.D., John J. Hochfilzer, M.D., Conrad J. Holmberg, M.D., Kenneth A. Phleps, M.D., Robert E. Priest, M.D., and George M. Tangen, M.D. 433 pages with index and 184 illustrations. Philadelphia and London: W. B. Saunders Co., 1949.

This is an excellent book for undergraduate students and as a reading text in postgraduate courses. Dr. Boies is well qualified to write such a book. He states the objectives of the book in his preface and these objectives are well fulfilled.

This volume should be in the library of all medical schools.

T. E. W.

The Comparative Anatomy and Physiology of the Larynx. By V. E. Negus, M.S., F.R.C.S., Surgeon to the Ear, Nose and Throat Department of King's College Hospital, London. With foreword by Sir Arthur Keith. First edition. 230 pages with index and 191 figures. New York: Grune & Stratton, Inc., 1949.

This book, as the author states in his preface, is a condensation of his previous work, "The Mechanism of the Larynx." It is an excellent and complete work, profusely and beautifully illustrated. The author leaves no phase of the anatomy or physiology of the larynx untouched.

Particularly valuable for the clinician is the chapter on the anatomy of the human larynx and the appendices. This book should be in the library of every department of laryngology. T. E. W.

The Nose. An Experimental Study of Reactions Within the Nose in Human Subjects During Varying Life Experiences. By Thomas H. Holmes, M.D.; Lester H. Hofheimer, Research Fellow in Medicine; Helen Goodell, B.S., Research Fellow in Medicine; Stewart Wolf, M.D., Associate Professor of Medicine; Harold G. Wolf, M.D., Professor of Medicine (Neurology), Cornell University Medical College, New York, N. Y. With a foreword by Warfield T. Longcope, M.D., Professor Emeritus of Medicine, The Johns Hopkins Medical School, Baltimore, Md. 154 pages with index and 37 illustrations. Springfield, Ill.: Charles C. Thomas, Publisher, 1950. Price $4.50.

This is a most interesting treatise on nasal pathologic changes resulting from situational threats. The subject is too frequently overlooked by rhinologists. Of particular interest and importance are the histologic and cytologic findings reported by the authors. The concept that eosinophiles in secretions and tissues is pathognomonic of allergy must, in the light of their observations, be seriously questioned.

The book makes fascinating reading and is strongly recommended to all otolaryngologists. T. E. W.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

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Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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THE
LARYNGOSCOPE.

VOL. LX JUNE, 1950. NO. 6

STRUCTURAL CHANGES IN THE CILIATED EPITHELIAL CELL DURING UPPER RESPIRATORY INFECTION: PRELIMINARY REPORT.

WILLIAM T. K. BRYAN, M.D.,
and
MARIAN PFINGSTEN BRYAN, A.B.,
St. Louis, Mo.

During the routine study of nasal smears we thought it would be interesting to stain the secretions of the common cold according to Dr. George N. Papanicolaou's method.[1,2] His stain has proved particularly suitable for epithelial cells in differentiating malignant changes. It seemed reasonable that such a stain would give additional information on changes which take place in the nasal epithelium during acute upper respiratory infections.

Nasal smears have been repeatedly studied with the more traditional stains, as Wright's, Giemsa's and Hansel's. In 1930 and 1934, *Hilding*[3] reported studies of the common cold in which he had observed large numbers of living epithelial cells and fragments of cells in the secretions during the first three days of a cold. In order to study the pathological changes he took biopsies from the noses of 30 volunteers suffering from colds in various stages. The most striking change was the extensive loss of epithelial cells, both of the columnar and of the deeper lying stellate types. After a few days the

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Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 15, 1950.

epithelium regenerated and resembled the normal controls. All specimens were stained for bacteria and carefully examined, but no bacteria were ever found to invade the epithelial cell.

The material for this study consists of numerous nasal smears taken at various times in the course of colds and of others taken regularly twice a day throughout the duration of the infection. To date four complete series and numerous incomplete series and isolated smears have been studied with Papanicolaou's stain, in all 75. Most of the smears were made by blowing the material from the nose, rather than by swabbing, and then spreading the material gently with a metal probe. Duplicate smears were made in each case in order to have a comparison with Wright's stain.

Ciliated epithelial cells appear in moderate numbers in the first smears taken, that is, within six hours of the onset of nasal secretion. Succeeding smears show an increasing number of these cells which reach a peak, depending upon the severity of symptoms, between the second and fifth days. The number then diminishes, the cells becoming very rare as the cold subsides.

The ciliated epithelial cells show definite degenerative changes in contrast to the normal ciliated epithelial cell.

Normal columnar ciliated cells appear in Fig. 1, obtained by swabbing from a normal nose. In the Papanicolaou stained smears the cilia are clearly and probably almost perfectly preserved—which is not true in smears stained with Wright's stain. The bluish-purple nucleus is finely granular and contains one to three nucleoli. There is a definite nuclear membrane. The nucleus is usually eccentrically oval and is placed at the narrow end of the cell away from the cilia. Normal ciliated bronchial epithelium with this stain is described in "The Cytological Diagnosis of Cancer" by the Vincent Memorial Hospital staff.[4] It appears very similar to the cells we have seen in nasal smears. The cytoplasm is lightly granular and palely acidophilic or basophilic in the normal cell. In well preserved cells the cilia are clearly visible at the broad end of the cell; often the pink staining ciliary bodies can be seen. The cellular borders are distinct in the single exfoliated cells, but when they are in clumps they tend to be obscured. At times the cytoplasm is very elongated.

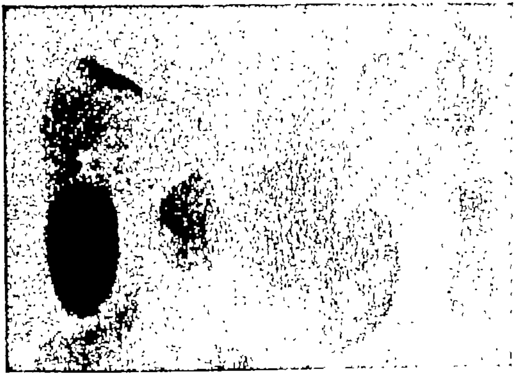

Fig. 1. A group of normal ciliated epithelial cells from a nasal smear.

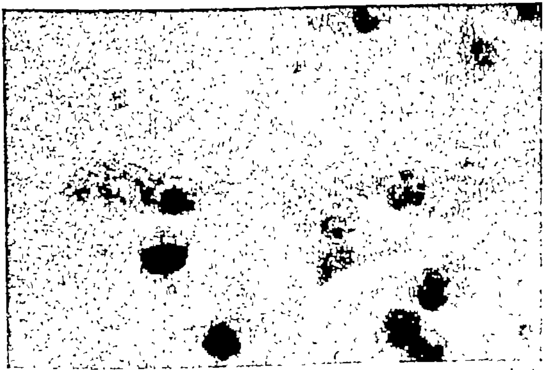

Fig. 2. Three degenerating ciliated cells from an acute upper respiratory infection. The left and middle cells have pyknotic nuclei, nuclear halo with two black bodies, and a pink cytoplasmic body. The cell on the right shows karyorrhexis. Magnification is about 950×.

Frequently normal goblet cells with ballooned vacuolated cytoplasm are identified. Squamous and basal epithelial cells are also normally identified and appear in varying numbers.

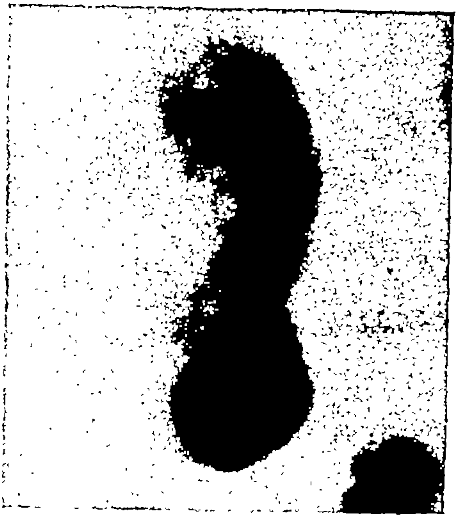

Fig. 3. Karyorrhexis with pink nuclear inclusion bodies (density in black and white is lighter than the masses of chromatin).

In contrast to the normal ciliated columnar exfoliated cell, the remaining illustrations show examples of degenerative changes which occur in these cells during a cold. The nuclear degeneration seems to fall into three groups according to the following patterns:

1. In the first there is a marginal arrangement of the basophilic chromatin on the nuclear membrane. The cytoplasm and cilia appear normal.

2. In the second the nucleus shows karyorrhexis with irregularly distributed pyknotic clumps of chromatin. These cells usually show a halo around the clumps. In between the dark masses of chromatin one or more pink bodies are occasionally found. The cytoplasm is more roughly granular and often contains one or more smooth, pink spherical bodies usually near the nucleus. The cilia appear normal. Some of the cells are elongated and constricted in the middle. This is more marked and numerous in the following pattern.

3. In the third the nucleus becomes completely pyknotic. Often it is seen as one solid, round, black mass. The eccentric position may or may not be maintained. About the chromatin mass there is frequently a rather clear halo which may contain one to three small black bodies more like granules of the same dark nuclear color. One is impressed with the frequency of this pattern. The smooth, pink spherical body or bodies in the cytoplasm mentioned in the second pattern are regularly present in this group. This group is most numerous in all cases thus far studied at the peak of the desquamation.

At the same time there are numerous "empty nuclei" in ciliated cells. These seem to have lost the black mass of chromatin material. There appears to be a clear area without visible structure. All manner of nuclear material including whole pyknotic nuclei, that is, the spherical black masses,

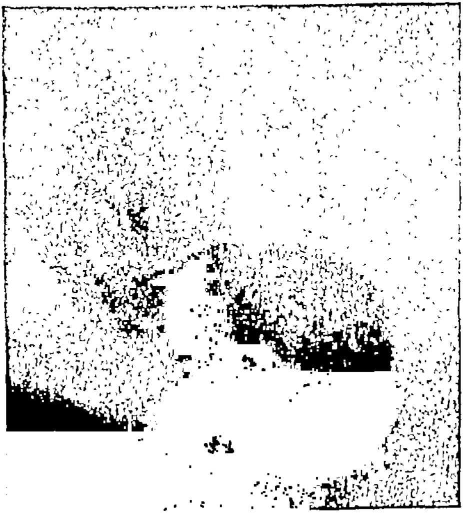

Fig. 4. Degenerating spherical cell. The ciliary bodies and cilia are quite prominent. Nucleus is pyknotic.

with or without halo and small black bodies, are seen scattered unattached over the slide.

The cytoplasm seems to fall into two patterns of degeneration. One is the shortening of the cell, some becoming almost spherical (see Fig. 4). The other pattern is of constriction

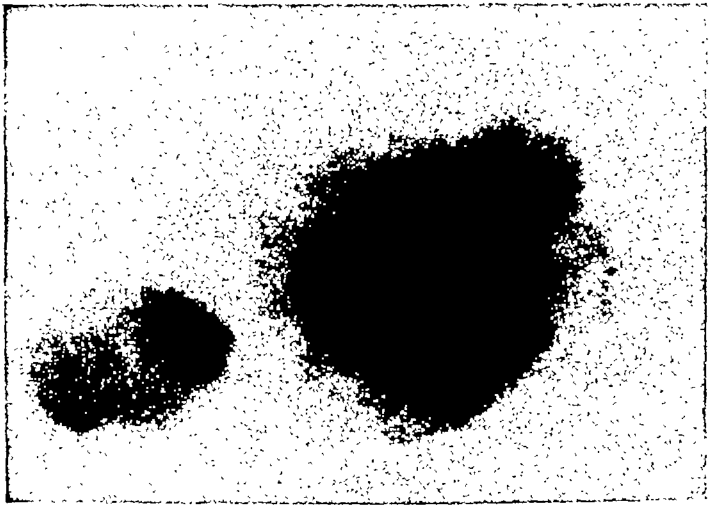

Fig. 5. Tufted ciliated cell. Nucleus shows karyorrhexis and pink body.

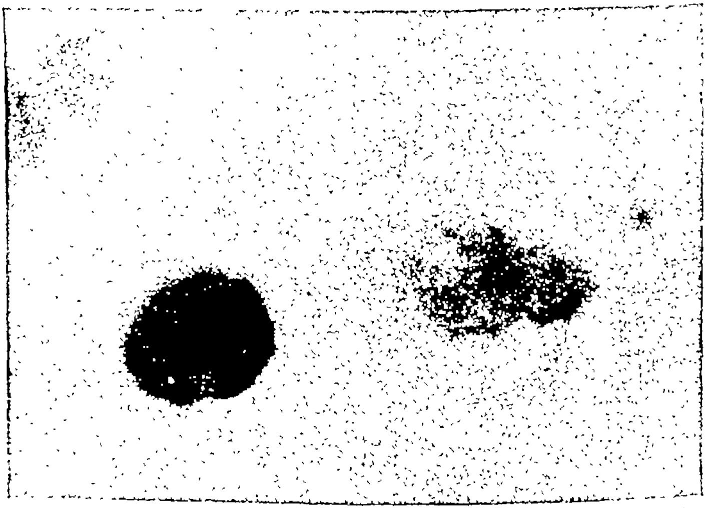

Fig. 6. Nuclear portion is beginning to separate and shows margination of chromatin.

and elongation, even to the point of breaking in half (see Fig. 6). The nucleus tends to be in one half, the bulk of the cytoplasm and cilia in the other. This seems to be a progressive process, as after these halves separate, many cellular

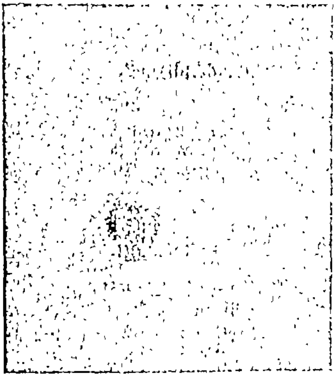

Fig. 7. Separation of nucleus and cytoplasmic portions is complete.

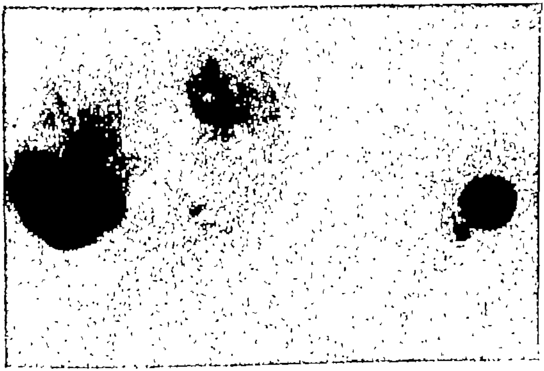

Fig. 8. Isolated nucleus to right. Two degenerating cells left showing karyorrhexis and center one margination.

fragments are present in the slide. Many tufts of cilia attached to small portions of cytoplasm without nucleus are seen. In both patterns the cilia may arise from the sides of the cell. This wide attachment of cilia has been observed in tissue cultures of ciliated epithelium.[5] In the more spherical they are seen attached to a large part of the surface. In these the nuclei appear more normal. The pink cytoplasmic body is found in the majority of all the degenerating ciliated cells.

In all these slides of acute colds very few bacteria are noted and they have never been seen within the ciliated epithelial cell or appearing to invade it. Evidently these epithelial cells degenerate even though the presence of bacteria is not evident.

DISCUSSION.

The Papanicolaou stain gives particularly clear nuclear detail which makes possible the determination of and the distinction between the various types and conditions of exfoliated epithelium; whereas Wright's, Giemsa's and Hansel's stains are especially good for showing eosinophiles. The epithelial cell is readily identified as such in these stains, but the nuclear detail and cilia are not clear;[3] nevertheless, in the smears stained lightly with Wright's one can identify with difficulty the same halo, pink and black nuclear and perinuclear bodies.

The consecutive series of smears are especially valuable in determining the timing of desquamation. In random or routine slides the epithelial cells were not always found in large numbers. The ciliated epithelial cells are rare in smears taken from subacute and chronic infections; but these structural modifications have been seen in all cases of acute upper respiratory infection thus far studied.

The stain is selective for the degenerating epithelial cells as they are easily spotted by their acidophilic reaction.

The significance of the intranuclear and cytoplasmic bodies, both pink and black, is unknown. Nevertheless, Dr. E. V. Cowdry and Dr. Walter Siebert, pathologist, have examined these slides and suggest a possible correlation of these changes with the virus etiology of a cold. The marked progressive degeneration of both nucleus and cytoplasm is evidence of injury to the cell.[6]

SUMMARY.

By means of the Papanicolaou stain, degenerative structural changes have been shown to be present in the exfoliated ciliated epithelial cells in nasal secretions. Prominent among these are intracellular bodies of two kinds which to our knowledge have not previously been described. Their identification and significance at this time awaits further investigation.

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THE FINDING OF POSITIONAL NYSTAGMUS IN THE DIZZY PATIENT.*

RUSSELL FLETCHER, M.D.,
Berkeley, Calif.

The complaint of severe, recurrent dizzy attacks that occur with a change of position of the head or body is very common in our practice. Positive clinical findings unfortunately are rarely found and the dizziness is attributed to a functional disorder or even malingering in those claiming dizziness following a head injury. Positional nystagmus, when found, is positive evidence of organic pathology that is causing the dizziness. It is easily seen, easy to test for and is an essential part of every complete otoneurological examination.

The primary purpose in presenting this paper is to describe positional nystagmus, and how to detect it in the dizzy patient. A relatively brief discussion will be given on the mechanism of positional nystagmus and the location of the pathology. Some of the interesting points and problems presented by these patients will be discussed. A movie of two of the patients will be shown.

Although we had a patient in 1935 who had severe positional nystagmus, no other cases came to my attention until two years ago. Since then I have seen 12 cases that showed very active positional nystagmus and presented problems that made it necessary for me to investigate the subject farther.

Cases that had vertigo and nystagmus when the position of their head was changed have been reported for many years. Bárány was one of the first to report cases as early as 1911.

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif., Jan. 21, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

Although there has been some general discussion of its occurrence under the heading of physiology, pathology or mechanism of nystagmus, until recent years there has been very little written specifically on the subject of positional nystagmus. The textbooks usually mention only the fact that a change of position or posture may cause vertigo or nystagmus. Positional nystagmus is not listed in the index in many textbooks.

Most of the neurosurgeons and otologists who were asked about positional nystagmus were not familiar with it. Last year at this Section Meeting in Los Angeles I asked several of the members about it, and as very few of them had a speaking acquaintance with the term positional nystagmus, it seemed to me that this was a very appropriate subject to present at this meeting.

HOW TO TEST FOR POSITIONAL NYSTAGMUS.

Positional nystagmus is the nystagmus that is produced when the position of the patient's head is changed or when the head is in a certain posture. As no other stimulation is required, the tests are, therefore, very easily and simply done. If the patient gives the history of this type of dizziness, it might be well to test for positional nystagmus before proceeding with the usual otoneurological examination, since this finding is not constant, as will be explained later. The patient is put on a couch or table. At first he sits erect and then lies straight back to the supine position. This may cause the nystagmus. In others it will occur if they lie back with their heads to the right or to the left. Although some writers seem to stress the possible effect of torsion of the neck as the cause of this, it is my personal opinion that it is not important. If this were true we would have it very frequently when we consider that 150,000,000 people twist their necks all day and night. Some patients while in the supine position will rotate their head to the right or to the left and have positional nystagmus. Some patients become dizzy and have nystagmus when they sit up from the supine position or put their head back in the optimum position from the head erect position.

Other positions that can be tried are the head hanging position or stooping over position. In other words, any change of position of the head may elicit nystagmus and some patients develop the nystagmus in several of these position changes.

Positional nystagmus is an abnormal pathological finding and is accompanied by severe vertigo, sweating, pallor, etc. It occurs spontaneously in patients who have no other illness and in patients following a head injury as well as in patients with CNS pathology, such as brain tumors, multiple sclerosis, etc. It frequently is the only pathology found (by neurologists or otologists) to account for dizziness. In all of my cases there were essentially normal findings in the ear, nose and throat, including the audiometer test, the Romberg test and the caloric and rotation tests. There was no spontaneous nystagmus or pastpointing in the head erect position.

The nystagmus that occurs in the cases I have seen is very active and wild and of greater amplitude than that produced by caloric or rotation tests. Frequently the patient will shout, "There it goes!" and will grab hold of anyone or anything available, and they appear very frightened. They may close their eyes, but the eyes can be seen to twitch violently beneath the lids. Sometimes the nystagmus does not start for several seconds after the change of position. It may last a few seconds or it may continue for minutes. Many patients have complained of dizziness, but unless they had active nystagmus for several seconds I have not called them true positive cases of positional nystagmus.

What kind of nystagmus do you see when they are active? It seems to me that my cases have shown every type there is—horizontal, rotatory and vertical. They are so active and wild that many times I have not been able to tell exactly in what direction the quick component moves. When I first started having movies taken of these cases, about a year ago, I had hoped to have high speed movies made in order to help me analyze just what their eyes were doing. I took six of my patients to the USN Hospital at Oakland to have movies taken in their photographic laboratory. The movies have been one disappointment after another. Either the patient could not be made to produce nystagmus after repeatedly changing his position or else the camera did not perform when the nystagmus was active — the camera was not wound up, it broke down or was out of focus, etc., at the crucial moment. Finally with the aid of Dr. John Howard, who has a new Bolex camera, and the help of a patient who performed regularly, we were able to get one good film. This film shows a definite vertical nystagmus, whereas my notes showed that I had considered it "marked rotatory nystagmus, with a vertical element." Having seen this movie, I am not sure that some of the other cases that I called rotatory would not have shown more of a vertical character in a movie.

Although the nystagmus may occur in any direction, or even may change direction during an attack, it is very important to detect the quality, character and direction of the nystagmus in order to interpret the mechanism of this phenomenon. This is particularly true if there is a vertical nystagmus, for we have always considered spontaneous vertical nystagmus to be of central origin.

When do you make the tests for positional nystagmus? Whenever the patient describes his dizziness as coming on when he changes his position, you should make tests for positional nystagmus. It must be very strongly emphasized here that you may have to repeat the test several times to elicit positional nystagmus. This was forcefully impressed upon me by my first patient who was sent to me by an insurance company before appearing before the Industrial Accident Commission. He had a fractured skull with unconsciousness 20 months before. Since his injury he complained of occasional severe dizziness when he moved his head. Two well known neurosurgeons had never found organic pathology to account for his dizziness. My otoneurological examination was completely negative. Once, when he put his head in the optimum position, he said he was dizzy, but there was no nystagmus. I had him lie down and change his position in various ways to make him dizzy. The following day I repeated the examination, which was again normal. I then explained to the patient that I could find nothing organic to account for his dizziness and although I did not doubt that he felt dizzy, I would have to send a report of normal findings to the insurance company. The patient remonstrated so loudly and sincerely, while I was trying to usher him out of the office, that I consented to test him once more. Again I had him recline from the erect sitting position to the supine position and he suddenly yelled, "There she goes!" He was right. He had a wild rotatory nystagmus to the right that lasted 15 seconds. Repeating this procedure twice produced no dizziness or nystagmus but the next attempt produced dizziness and nystagmus. The necessity for repeated testing has also been impressed on me in my attempt to get movies of these patients, as previously discussed. You do *not* get positional nystagmus every time you test the patient.

It should be emphasized that if positional nystagmus is found, it is positive evidence of organic pathology that is causing the vertigo and it eliminates the diagnosis of functional basis for the dizziness. This is very important for all patients, and particularly important for the post-traumatic industrial case. If you do not find nystagmus it does not necessarily mean that the patient has not had, nor will not have, positional nystagmus; also, it should be emphatically stated that any otologist who does not make the test for positional nystagmus on two or three occasions on a patient who says he is dizzy with a change of position is not making a complete otoneurological examination. I am emphasizing the necessity of careful testing, repeated if necessary, for I am sure the chief reason that we all have failed to see more of these cases is the fact that we have not tested sufficiently for it.

THE MECHANISM OF POSITIONAL NYSTAGMUS.

Bárány proposed the theory that the otolith system was concerned with static equilibrium, vertigo and nystagmus, and felt that positional nystagmus was due to disturbance in the otoliths. A great deal of research has been done and many articles written upon the entire subject of the complicated mechanism of nystagmus. The utricle was attributed by many to be the site of origin of positional nystagmus. As you all know, there is a considerable difference of opinion on many aspects of nystagmus. It seems to me that it is not advisable to enter into this controversy at this time. Instead of this I am going to give two references that I feel are outstanding, and I am going to quote briefly from each, for I believe that they bring this subject up to date in the best possible manner.

The first reference is to McNally's article entitled, "The Physiology of the Vestibular Mechanism in Relation to Vertigo."[1] After reviewing the literature and experimental work, McNally said: "Such well controlled experiments as these do not indicate that nystagmus arises from utricular stimulation." McNally also said: "Nylen and Lindsay have classified positional nystagmus into type I and type II. In type I, the direction of the nystagmus changes with a change of position of the head. It occurs predominantly in CNS lesions. In type II, the direction of the nystagmus does *not* change with changes in position of the head; but the nystagmus may appear only in certain positions or it may be influenced in intensity by the position of the head. It may be found *either* in peripheral lesions or in central lesions." "It has not been *definitely determined whether the cause of positional nystagmus* is a lesion within the labyrinthine end organs or whether it is the result of some lesion within the CNS."

The other important reference is to the papers by Dr. J. R. Lindsay.[2,3] He writes: "Observations on known central lesions such as tumors, multiple sclerosis and cerebral concussion agree that a positional nystagmus type I invariably indicates a central location, whereas type II may occur either in a peripheral or central lesion." "Positional nystagmus is a relatively infrequent occurrence in lesions known to be situated in the peripheral ear." "It was demonstrated infrequently in the Ménière's disease group."

My own opinion is that positional nystagmus is usually due to central pathway pathology. The vertical nystagmus frequently seen in these cases and shown in the movies seems to be strong evidence of this. The fact that the hearing tests, caloric tests and rotation tests are normal in most of these cases does not point to a peripheral lesion. Unless there is evidence of peripheral vestibular pathology and until the question is definitely settled as to the location of the lesion that causes positional nystagmus, the patient should be given the benefit of the doubt and be given a thorough neurological examination to search for any CNS pathology.

The following are a few remarks about my cases that may bring out some points and problems not already mentioned: my first patient, as previously mentioned, emphasized to me that repeated tests may be necessary to elicit positional nystagmus. This positive evidence of an organic basis for his dizziness, which had not been detected before, was very important in his disability rating. He was the first of five cases following a head injury. Disability, industrial medicine and medicolegal aspects are a big problem in these cases.

One patient was 70 years old and had no other pathology than her positional nystagmus. Her dizziness stopped within a month. Pyribenzamin did not help her dizziness.

Mrs. B., age 34, was in the U.S.N. Hospital for weeks under investigation. Her first dizzy attack came on during pregnancy several years before. She had had several series of attacks. No one had noted any nystagmus previously and she was beginning to wonder if she were imagining it. She had completely negative findings except the positional nystagmus, which could be produced very regularly whenever she moved from the erect to the supine position. It continued for a month. We took movies that were not good enough to show at this time. With the last series of attacks she had very severe occipital headaches. No treatment, including Dramamine, helped her dizziness, although it did help her nausea and vomiting. Dramamine did not help the dizziness in three other patients and made one worse.

Dr. Howard, an intern at Oak Knoll Hospital, had some dental work done with his head far extended and some heavy pounding on his teeth the morning of onset. His dizziness lasted about a month. He had one very remarkable observation: He is an excellent swimmer and during this time he noted that when he was swimming under water and made a turn he became dizzy and he had complete loss of his orientation as to whether he was swimming deeper or toward the surface. This scared him so that he requested the lifeguard to watch him when he swam under water. Such a loss of orientation might be disastrous to an aviator.

A Ford Motor employee had a fractured skull in July, 1948, and was under the care of very well known neurosurgeons who had found no organic reason for his dizziness when he changed the position of his head. Marked positional nystagmus was found, after several attempts, when he went from the erect to the supine position. The question has repeatedly come up as to his ability to work and his prognosis. At the present time he does not show positional nystagmus, but he does say that he is dizzy when he moves his head. Any patient who has postural vertigo and positional nystagmus is surely not able to work as a carpenter or a structural steel worker where falling might result and cause further injury. The prognosis of these patients is indefinite. Most cases seem to clear up in time, although some have recurrent attacks over a period of years. Apparently they do not have a progressive, degenerative process, although they may have recurrences.

Another industrial and medicolegal case had a severe head injury and had terrific nystagmus when he went from the sitting to the supine position. Following the caloric tests, which were normal, the patient did not have dizziness and nystagmus when tested and has had none since.

Mrs. G. had a slight spontaneous one degree rotatory nystagmus to the right. The caloric tests were essentially equal in both ears. When the patient went from the upright to the supine position the spontaneous nystagmus stopped for a few seconds and then became two degrees rotatory. Dramamine relieved the nausea but not the dizziness.

One patient who had positional nystagmus when seen once at home went to Lane Hospital for further observation. They reported a marked decrease in the caloric reactions on one

4. Although the tests are very simple to perform, frequently it is necessary to repeat the test several times on two or three occasions in order to elicit the positional nystagmus which is very marked.

5. An otoneurological examination is not complete unless these tests have been performed on every patient who complains of dizziness that occurs with a change of position of the head or body.

6. Although it has not been definitely settled as to whether the cause of positional nystagmus is in the peripheral or central vestibular mechanism, it is the present conception that most of these cases are of central origin. This is brought out by the fact that the hearing tests, caloric tests and rotation tests are normal in many of these cases, as well as the fact that their nystagmus is frequently vertical in character.

7. Most of these patients get well spontaneously. No medication seems to effect the dizziness. Although the patients may have recurrent attacks of dizziness, the pathology apparently is not a progressive degenerative process, unless the pathology is due to a lesion such as a tumor, multiple sclerosis, etc.

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2298 Durant Avenue.

EXTERNAL OTITIS.

IV — Cytologic Study of Secretions.*†

BEN H. SENTURIA, M.D.; JOHN I. MATTHEWS, M.D.,
and BENARD C. ADLER, M.D.,
St. Louis, Mo.

The present study was undertaken in an effort to understand the significance of the cytological components of secretions found in certain inflammatory diseases of the external auditory canal. It was thought that a study of the various normal and pathological cellular elements found in ear secretions might contribute to an understanding of the pathogenesis of external otitis and aid in the differential diagnosis of disease of the middle and external ear.

While bacteriological studies of secretions found in the external auditory canal have been numerous,[1-5] only an occasional reference has been made to the cellular components of the secretions. Politzer[6] examined the scales in acute diffuse external otitis and found large numbers of "micrococci." Proetz[7] examined the secretion following paracentesis in a case of allergic middle ear disease and noted cocci, a few leucocytes and epithelial cells, and an absence of eosinophiles. Recently Koch[8] reported an exhaustive study of the cytology of the secretions found in chronic otitis media and mastoiditis. He noted neutrophiles and bacteria in all discharges of chronic middle ear disease but did not report examinations of secretions of external otitis. Special emphasis was given to the presence of eosinophiles in allergic middle ear disease. Ts'en,[9] in 1926, studied the discharge from seven cases of acute and chronic middle ear infections. It was concluded

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*This investigation was aided by a grant from the Research Study Club of Los Angeles, Calif.

†From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 6, 1950.

that neutrophiles were present in both acute and chronic middle ear disease, but that in the chronic cases the neutrophiles were more apt to be degenerate forms. Dean and Pfingsten[10] while investigating the cholesterol content of various aural and nasal discharges also noted certain cytological features. They examined two cases of external otitis and noted epithelial cells and bacteria. Twenty-six cases of chronic otitis media and nine cases of acute otitis media were described, the discharges of which all contained neutrophiles.

Methods: The ear canals were cleansed of the mass of exudate by cotton swab or were irrigated with hypertonic saline,[11] and a smear was made of the exudate found in the sulcus of the canal. In cases of circumscribed external otitis the material was taken from the site of the discharge after spontaneous drainage or incision. The secretion was placed on the slide by rolling the applicator or very gently sweeping it across the slide so as to minimize morphological distortion of the cells. The smears were fixed by flaming and stained by a polychrome method (Wright's or two-stage eosin-methylene blue stain). In most instances a single smear was obtained prior to treatment. Some cases were followed by smears taken during the course of treatment in order to observe the cytological changes. A clinical diagnosis was recorded routinely prior to the inspection of the slide, although in a few cases the original diagnosis was revised in view of subsequent findings and the course of the disease.

The inflammatory diseases of the ear were divided into the following categories according to their clinical findings.[12] A small number of normals were included as a control.

A. Normal

B. External Otitis
  1. Acute diffuse
  2. Chronic diffuse
  3. Circumscribed
  4. Otomycosis
  5. Seborrheic
  6. Neurogenic

C. Otitis Media

D. Postoperative Mastoidectomy and Fenestration.

Smears obtained from ear discharge were examined for the presence of the following elements and the numbers of each of these were graded from zero to four plus:

Neutrophiles
Lymphocytes
Eosinophiles
Bacteria
Epithelial cells
Erythrocytes
Mucus strands*
Fungi.

Epithelial cells were classified according to shape, the presence or absence of a nucleus and the staining characteristics of the cell. The dispersion of the epithelial cells as singly, or in sheets or clumps was recorded. An effort was made to determine if there was bacterial invasion of the epithelial cells, but differentiation between true invasion of the cells and superimposed bacteria was difficult.

RESULTS:

Examination of the smears revealed certain general features. A nucleus was not visualized in more than half of the epithelial cells. It was found that no apparent significance could be attached to shapes of epithelial cells since in any single slide one would find a wide variety of shapes and arrangements of the cells. Eosinophiles were rarely found. Red blood cells were noted only in smears taken following a myringotomy or an incision of a circumscribed abscess.

The findings on smears in the various clinical categories as summarized in Table 1 are as follows:

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*These were bluish or purplish staining filamentous structures varying markedly in size from 20 to 100 mu in length and 2 to 10 mu in width.

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HEMORRHAGE IN EAR, NOSE AND THROAT.*

LELAND G. HUNNICUTT, M.D.,
Pasadena, Calif.

A simple nose bleed does not cause us to worry, but all of us know there are very difficult hemorrhage cases in which we wished we knew one or two tricks to solve the problem. This discussion will bring out procedures, many of which have been passed on by men like you and, if they are new to you, will prove to be of value when the difficult hemorrhage case suddenly appears. Suffice it to say that almost every known cause of hemorrhage can appear in our field and almost every part is subject to hemorrhage. Knowing this, we should then be acquainted with the different means of controlling the hemorrhage. No attempt will be made to give a textbook coverage of the subject, such as giving the detailed blood anatomy and all the causes of hemorrhage in our field.

Epistaxis is most frequently encountered by us in the age group under 50 years. Each one has worked out a satisfactory method of stopping bleeding from the anterior part of the *septum in children when the bleeding is brisk, but when there* is just a little bleeding from both sides and a large area for a long period of time it presents a problem. Providing general causes have been eliminated, it can be controlled or cured in most cases by having the child wear a light plug of cotton in the nostrils for about 15 minutes at a time, two or three times a day. This follows Hilding's observations that shutting off the air in a nostril will allow the tissues to return to normal when the protective mucous sheath is present.

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif., Jan. 21, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

Bleeding from the adenoid while the child is still in surgery may be controlled, other than by applying pressure from a string sponge, by applying the raw surface of a tonsil which has just been removed. It is first necessary to locate well the point of bleeding, then grasping the tonsil with an Allis clamp, press firmly over the bleeding point for about two minutes. This was first done on the basis of what most of us have done when a lateral sinus is bleeding, and the application of a piece of adjacent connective tissue, or better yet, a piece of muscle will stop the bleeding. The observation that vomitus tends to stop bleeding from the tonsil and adenoid area has suggested that dilute HCl be used. It is diluted down to about one-third strength, a sponge is soaked with it and pressure applied.

Bleeding from a hidden place in the nose in one under 50 years of age, and also to be found over 50 years of age, is often from the artery going to the inferior turbinate. A quick and efficient method of stopping the bleeding is to crush the turbinate over its entire length. It should be noted the the artery is at the surface on the lateral and inferior aspect just as the artery enters the turbinate at its posterior end, and it is at this point that the crushing does the work. The technique was suggested by a 30-year-old female patient who was quite exhausted after a week of attempting to stop the bleeding by the usual means, including postnasal and nasal packs, along with a number of transfusions. She suggested that a careful check on the blood supply be made and note which artery was nearest the surface. Her satisfaction was considerable when the technique about to be described stopped the bleeding. The nose is freed from clotted blood. An applicator with saturated cocaine solution is rubbed below and above the inferior turbinate from front to back. Novocaine is injected at the anterior end, at the mucocutaneous junction. A curved clamp without teeth at the end, and with blades long enough to include the entire length of the turbinate, is placed with the convexity outward along the attachment of the inferior turbinate. The clamp is closed tightly on the turbinate and left in position for a half minute before removal. If successful, the bleeding stops and the turbinate shrivels up, due to its avascular state. No packing is necessary, no discomfort follows and in a week the turbinate has resumed its former appearance. Do not turn the turbinate upward or the artery will be torn from its moorings and the bleeding continues. Quite a number of hemorrhages in people over 50 years of age come from this source, and this method is quite satisfactory in that it can be done easily in the home.

If bleeding is not controlled, then a 5 cc. Foley catheter is inflated with water and used as a postnasal plug. If fortunate enough to make contact on the bleeding artery, it is entirely satisfactory and quite comfortable. The usual methods of gauze packing, both nasal and postnasal, are known to all, so will be passed over.

When bleeding continues, especially in one over 50, the electric cautery is used. The blood usually comes from the sphenopalatine artery near the posterior end of the middle turbinate. The patient is put in the hospital, given an intravenous pentathol anesthesia while in the head low position. Sitting at the head makes it easy for the operator. The palate is elevated by two catheters. A metal suction tip covered with rubber is passed through the nose and is guided by viewing through a large laryngeal mirror. It may be necessary to bend the end of the suction tip to make contact with the bleeding point. If good contact is made, the bleeding stops because it is all aspirated into the tube. The electric current is turned on and the tissues coagulated. The "cutting" and not the "coagulating" current is used because the "cutting" current is more superficial, thus resulting in less sloughing. The amount of current must be determined by each operator at the time. A note should be made in regard to the suction tube. Use a rubber tube small enough so that it will cling tightly to the metal tube, leaving only about an eighth inch of exposed tip. By first putting a few drops of oil in the rubber tube it will easily slide on the metal tube.

Considerable trouble in getting down to the external carotid artery has at times proved embarrassing, not only to some of us but to general surgeons when it was hoped he could do it more efficiently. Following Dr. Barnhill's technique of blunt dissection and practicing on monkeys, it was found there was little difficulty in quickly exposing any desired vessel in the carotid sheath area. To review quickly, a skin incision is made just anterior to the sternomastoid, the fascia is nicked and the fascial layers are separated by inserting two Kelly hemostats, and gripping them firmly, the tissues are separated for the length of the incision. Use little or no retraction as the landmarks become distorted. It may be necessary to again nick the fascia before inserting the hemostats for another separation. By this blunt dissection, there is little or no bleeding.

Once in a while it is necessary to ligate the ethmoid arteries. This is done by the external route, and upon exposing them, instead of ligation the use of the "cutting" current works well. This was thought to be a procedure without danger until one of our members had a case in which blindness followed the cauterization of the posterior ethmoid artery. If heat from the cautery was the cause, then ligation is to be preferred.

Bleeding from the ear has been encountered mostly from blebs. When blebs are seen before they have ruptured, a quick way to drain them is to instill 25 per cent magnesium sulphase in glycerine. It is quite painful, so the patient is given something for pain before the treatment. In a few minutes the blebs either rupture or flatten out. The magnesium sulphate dissolves with difficulty. A warmed mortar and pestle (about 110°) is used and the magnesium sulphate is ground into the glycerine over a period of about two hours. Only occasional grinding is necessary.

Bleeding from the lungs or esophagus is of interest only to the endoscopist and will be passed over lightly. A good internist or chest man is quite necessary to give aid in proper diagnosis and treatment. Seldom is a bleeding point found on bronchoscopy as the bleeding comes from farther down. A technique has been described for plugging a bronchus by heavy cautery, and in this way the bleeding is dammed up in the lung below. Bleeding from the esophagus may require treatment through the esophagoscope or the aid of a surgeon may be necessary. No personal experience of value can be passed on to you.

Recently Dicumarol has been used in treating multiple sclerosis, and there is constant danger of lowering the prothrombin time to the point of spontaneous hemorrhage throughout the body. Such a case was recently encountered and since other cases are likely to occur, it is well to relate our experience. There was an emergency call to do a tracheotomy on an almost unconscious 30-year-old female who was on a maintenance dose of 50 mg. of Dicumarol. Examination showed a submucosal hematoma of the posterior pharyngeal wall, so large that it pressed against the tongue. By the time the patient arrived in surgery breathing had stopped and a quick tracheotomy was done, after having injected novocaine with adrenalin. In the meantime the prothrombin time was found to be zero. A blood transfusion had been ordered. Within about 10 minutes after the tracheotomy was done hemorrhaging began so profusely that it could not be aspirated fast enough. The patient was turned over for postural drainage and the transfusion was started. Finally two blood and one plasma transfusions were going in three different veins. After about an hour the bleeding stopped and a blood pressure could be obtained for the first time since surgery. The systolic was 40. Looking back, there seems to be little else that could have been done, but this serves as a warning to us that we have many potential severe hemorrhage cases in those patients who are going along on maintenance doses of Dicumarol.

Many of you have in mind other methods of controlling hemorrhage, such as the use of Gelfoam and similar preparations; salt fat pork, as told to me first by Dr. Arthur Jones; crisscross incision and Sylnasol injection for bleeding from the anterior part of the septum as experienced by Dr. J. Mackenzie Brown; the use of sea sponges as described by Dr. Gilbert Roberts, in which they are used not only for splinting but for controlled bleeding as well; injection of novocaine with adrenalin in the tonsil bed for any stage of post-tonsillectomy bleeding as described to the author by Robert Odom, and many others could be related.

It is hoped there will be a free exchange of ideas so that we may all profit by the other man's experience. We should make an effort to know well the methods of controlling our most difficult hemorrhage problems and there will certainly be less apprehension and time wasted when the emergency arises.

THE MANAGEMENT OF NASAL FRACTURES.*

GILBERT ROBERTS, M.D.,
Pomona, Calif.

Nasal fractures, like tonsils, apparently have come to be regarded by the otorhinolaryngologist as a subject too elementary to warrant further study or discussion.

A review of the literature for the past 10 years appearing in the American journals devoted to otorhinolaryngology revealed a voluminous bibliography on rhinoplasty but only a half dozen noteworthy articles on the management of nasal fractures.[1-6]

Even the standard textbooks on otorhinolaryngology have passed hurriedly over this subject with few or no details as to technique.

With the rhinologist entering the field of rhinoplasty, it seems strange that one of the basic causes of the need for rhinoplasty, as well as for submucous resection, should receive so little attention.

It would appear that the rhinologist, interested in doing a better job of setting a broken nose, will have to refer to texts on plastic surgery and rhinoplasty in order to find the answer.[7,8]

It is unfortunate but true that nasal fractures all too frequently are dealt with hurriedly and inadequately, leaving the patient a result far from satisfactory cosmetically and, more important, functionally. In fact, many patients are told at the time that a corrective operation will be necessary later.

It is the purpose of this presentation to emphasize the need for a clear understanding of the anatomy of the external nose,

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Los Angeles, Calif, Jan. 22, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 30, 1950.

particularly the relationship between the bony arch and the cartilaginous structures and the importance of the latter in the end-result of nasal trauma.

Second, a technique permitting deliberate manipulation under adequate anesthesia, followed by adequate splinting, inside and out, will be described.

The framework of the nose is composed of a bony and a cartilaginous portion. The former is made up of the paired nasal bones, fused together in the midline, and articulating with the nasal process of the frontal bones superiorly, and the maxilla laterally. The strongest portion of this bony arch is at its attachment to the frontal bone, while its weakest and most frequently fractured portions are the free margins of the nasal bones inferiorly, and their attachments to the maxilla laterally; however, the bony arch is only half the picture, although all too frequently it is all that receives attention during hurried reduction.

The upper lateral cartilages, fused to the dorsal border of the septum, are loosely attached to the lower border of the nasal bones by means of fibrous tissue. Their intimate connection with the septum results in a guy rope action, with the septum acting as a ridgepole. Any forcible displacement of the septum may be followed by a tear in their fibrous attachment, with a lengthening of one guy rope and a shortening of the other, and a resulting deformity of the cartilaginous dorsum.

The septal cartilage, which rests in a groove in the vomer along the floor of the nose, has a doubly important rôle. It divides the nose into two air chambers, and anything that changes its position will alter the breathing function. Second, as mentioned previously, it serves as a ridgepole, guyed by the upper lateral cartilages.

The displacement of the septal cartilage from its groove in the vomer is a common occurrence; it has been shown to result from trauma at birth and from frequent falls and blows during childhood. The resulting deformity produces nasal obstruction, drooping of the tip and sagging and angulation of the cartilaginous dorsum. If treated early before fibrous union takes place, it can readily be corrected with thumb and forefinger, or with an Asch or Walsham septal forceps under light ethyl chloride or Vinethene anesthesia. If the bony dorsum has not been fractured, and it frequently is not, this is usually all that is necessary.

Injuries in adolescent and adult life, however, present a problem that requires a carefully planned and worked out technique. First of all, the rhinologist should not allow himself to be rushed into a hurried, spur-of-the-moment attempt at reduction under unfavorable circumstances.

We found from a large number of cases in the Navy that reduction could be delayed up to a week; and in cases of massive swelling and hematoma it was far better to wait until we could see what we were dealing with. X-ray films were not particularly helpful except in cases of depressed marginal fracture, which a soft tissue lateral view will show in the absence of clinical findings.

Lateral deviations and dorsal depressions should be looked for carefully, remembering that only the cartilaginous dorsum may be disturbed. If so, crepitus may be absent and point tenderness slight. Careful palpation may reveal a depressed fracture on one side only, at the junction of the nasal bone with the frontal process of the maxilla, without any apparent deformity because the dorsum is not shifted and the swelling hides the depression. Fractures of this type may produce a most surprising and disturbing deformity when the swelling subsides.

Finally, the nasal mucosa should be thoroughly shrunken with a spray of 10 per cent cocaine and adrenalin, all clots and secretions removed with spot suction, and the septum carefully inspected. Misalignment may be due to an old deformity or the result of the present injury. The presence of hematoma would suggest the latter; also, the septum may be found to be movable after intranasal anesthesia has been accomplished by sphenopalatine and anterior nasal nerve block with cocaine applicators.

If it has been determined that reduction is necessary, external anesthesia is accomplished by 2 per cent procaine nerve block as follows:

One centimeter above the inner canthus on each side a small wheal is made with a 26 gauge needle. Then a 22 gauge needle is inserted along the medial orbital wall to a depth of 2 cm. and 1 cc. of solution is injected. This anesthetizes the supra- and infratrochlear nerves which supply the upper part of the nose and also the anterior ethmoid nerves which supply the anterior superior portion of the septum. The infraorbital nerves are blocked by similar wheals 1 cm. lateral to the ala nasi, followed by injections into the infraorbital foramina. This anesthetizes the lower part of the external nose. This technique avoids infiltrating the tissues overlying the framework of the nose, and preserves an undistorted field.

Only two instruments are necessary for the ensuing manipulations: a narrow Lagenbeck periosteal elevator with a slightly curved tip is used to elevate depressed fragments; a Walsham septal forceps, which is similar to an Asch forceps, but with wider blades which cannot be completely closed, is the ideal instrument to rotate and unlock fragments and to realign the septum. One must learn to visualize mentally the position of the fragments and to note whether or not they are locked in order to realign them without undue force or further comminution; however, realignment of the septum may require considerable leverage, and frequently a lateral displacement of the dorsum which has not responded to manipulation of the bony arch will suddenly snap into alignment as the septum is straightened. A depression of the cartilaginous dorsum also will be corrected by elevation of the septum back into its groove in the vomer.

All these manipulations can be carried out unhurriedly and carefully with a minimum of bleeding and discomfort under the described anesthesia. One may take as long as 30 minutes to accomplish a desired result without undue complaint from the patient. Premedication with a hypnotic barbiturate is desirable, but morphine or a similar narcotic is rarely necessary.

After the manipulations have produced a satisfactory result cosmetically and functionally, the nose is splinted inside with foam rubber sponge material. This can be obtained at any 10 cent store in the form of powder puffs which can be sterilized by boiling. Cut in two, they require very little additional shaping to fit high in the nasal vault. Rolled or folded, they are inserted through the anterior nares, and they promptly spring back into shape, filling the nasal cavity much more satisfactorily than a Simpson splint. They exert a smooth, even pressure and stay in place without anchoring. It is the author's belief that they reduce intranasal swelling and hematoma materially and that they soak up secretions "like a sponge."

The external splint prevents swelling and hematoma, and holds bony fragments and torn cartilages in place until healing is well along. Soft lead or copper splints are advocated by many otorhinolaryngologists, while the rhinoplastic surgeons prefer dental stent; however, believing the former too difficult to shape and hold in place and the latter too heavy and cumbersome, the author finally decided to emulate the orthopedists and has been using a plaster dressing with excellent results. Before the plaster is applied, the eyes are protected with a few drops of mineral oil or olive oil. Then a cotton flannel butterfly is shaped to the nose and forehead and small strips of J & J Specialist Plaster, lightly soaked, are applied layer on layer, longitudinally and transversely, until a sufficiently heavy spica is built up. This is allowed to dry, which it does rather quickly, and then strapped snugly in place with adhesive strips across the forehead and cheeks.

The sponges are left in place five days, the patient receiving procaine-penicillin daily, and the plaster dressing is removed after 10 days.

In the author's experience the improved results of the above technique have more than made up for the extra effort involved, and while perhaps not as remunerative as submucous resection or rhinoplasty, have been a source of great personal satisfaction.

SUMMARY.

Nasal fractures all too frequently are dealt with hurriedly and inadequately, leaving an unsatisfactory cosmetic and functional result.

A clear understanding of the anatomy of the external nose, particularly the relationship between the bony nasal arch and the cartilaginous structures, is essential to the successful management of nasal fractures.

A technique permitting deliberate manipulation under adequate anesthesia, followed by adequate splinting, inside and out, is described.

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REHABILITATION OF THE PRESCHOOL DEAF CHILD.*†

DONALD K. LEWIS, M.D. (by invitation),
Boston, Mass.

The purpose of this paper is to present the experience of a modest clinical research program, a program designed for the rehabilitation of the preschool deaf child in a metropolitan area where virtually no other facilities exist. It is with reference to such areas of limited resources that I direct my remarks to you as otologists, rather than to teachers of the deaf or to those already participating in more complete programs.

In 1944, Dr. R. P. Guilder[1] reported preliminary results of an experimental program which offered intensive remedial assistance to a small, select group. This initial work was of such significance as to demonstrate the need for an expanded program. Dr. Schall and Dr. Meltzer, cognizant of the need for a closer alliance between the surgical and nonmedical therapy of deafness, gave us the opportunity of continuing and expanding this program. It is now an integral part of our plan for resident training at the Massachusetts Eye and Ear Infirmary, concomitant with the study of otosclerosis and fenestration technique. Its effectiveness is based upon the six following principals:

1. Diagnosis.
2. Estimate of Hearing Capacity.
3. Parent Education.
4. *Early* Application of Remedial Measures.
5. Continuous and Sympathetic Follow-Up Program.
6. Flexibility of Program as Needed.

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological ond Otological Society, Inc., New York City, N. Y., Jan. 5, 1950.

†From the Winthrop Foundation at the Massachusetts Eye and Ear Infirmary.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 27, 1950.

DIAGNOSIS.

The diagnosis of deafness in this age group may be confused with any of the conditions which cause a failure of language development. They are motor speech delay, developmental word deafness, mental retardation, lack of motivation and emotional traumata.

The following developmental language disabilities are an amorphous group, familial in tendency, associated with a disturbance of dominance in handedness and lacking in organic brain pathology.[3]

1. Developmental Word Deafness.
2. Motor Speech Delay.
3. Specific Reading Disability (Alexia).
4. Special Writing Disability (Agraphia).
5. Stuttering.
6. Developmental Apraxia (Clumsiness).

Stuttering, apraxia, and reading and writing disabilities present no problem as regards the differential diagnosis of deafness. They are frequently present, however, in combination with one of the other syndromes more easily mistaken for deafness. Motor speech delay is frequently seen to coincide with efforts to train a child away from a preferred left-handedness. Children with this disorder are attentive to and understand the spoken word. Most of them eventually develop speech to a varying degree of success. With their ability to understand speech they appear intelligent and adaptable.

Developmental word deafness, which would coincide with a pure sensory aphasia of the adult, is lack of understanding of the heard word. This interference of the receptive mechanism causes a delay and a distortion of speech, and prevents the acquisition of all these concepts formulated by the spoken word. The deaf child somehow manages to acquire these verbal concepts and subsequently his retardation is not so severe. The child with developmental word deafness is, at first, completely inattentive to speech and his early environmental investigation is limited to the medium of vision and touch. Gradually he begins to "echo" monosyllables, and with increasing maturity he learns the meaning of single words and may acquire an appreciation of very simple sentence structure with, however, errors in both pronunciation and grammar. Thus from the standpoint of symptomatology and therapy, he is an aphasic; but he is not classified as such because of his positive family deviation in handedness and absence of organic brain pathology.

The child with borderline intelligence, whose appearance may pass for normal and who may not bear the obvious stigmata of retardation, is frequently believed to be deaf, primarily because of his poor language behavior; so again his performance in nonverbal tests becomes important. The most constant finding to me in establishing a tentative diagnosis of retardation is the presence of what might be called "visual inattentiveness." The deaf child with his normal personal-social behavior is interested in people. He watches people, their movements, and is sensitive to facial expressions and often attentive. The retarded child may have a compulsive interest in objects, but is emotionally and intellectually unresponsive to people. He is prone to show poor muscular coordination, manual clumsiness and stereotyped purposeless movements.

Emotional disturbances, particularly those due to parental overprotection, are causative factors in speech delay. The child of oversolicitous parents, with his needs constantly anticipated, will lack the incentive for developing speech. At the other extreme, the institutionalized child without the give and take of ordinary home life develops a poverty of self-expression.

Etiology. A knowledge of the etiological factor responsible for the deafness may be helpful in estimating the hearing capacity and in predicting a tendency toward progression. In the order of frequency of occurrence in these patients, I have classified the deafness as congenital, infectious, hereditary and traumatic. The term congenital has been restricted to mean those who were born with defective hearing without any evidence of birth trauma or significant family history. Inherited deafness, present at birth, has been classed as hereditary rather than congenital. The child with a strong family history of inherited deafness is faced with the statistical probability of a further loss of hearing (see Fig. 1). I have not noted this retrogressive tendency in the congenitally deaf group where the deafness was the result of maternal rubella or erythroblastosis fetalis; furthermore, the rubella cases have almost invariably shown a correlation between the severity of the hearing loss and the month of maternal infection. Rubella occurring in the first month of pregnancy has resulted in a profound deafness in the offspring, while the deafness resulting from infection in the third month of pregnancy has not been so severe.

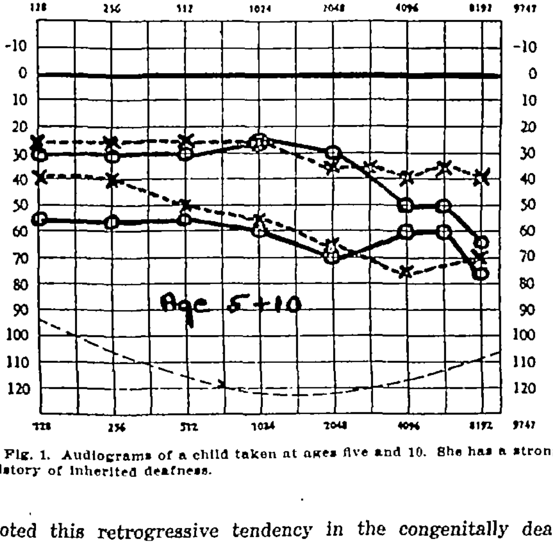

FIG. 1. Audiograms of a child taken at ages five and 10. She has a strong history of inherited deafness.

PARENTAL EDUCATION.

1. Personal discussion with physician.
2. Reading material.
3. Group conferences, lectures and social activities.
4. Follow-up interviews with social service.
5. Correspondence courses and instructive manuals.
6. Attendance at classes for speech and auditory training.

The medical consultations should clarify in the minds of parents the nature and degree of their child's hearing loss and should further emphasize the futility of a circuitous search for curative treatment which all too often results in such ill-considered procedures as prolonged vitamin therapy, radium treatments and unwarranted attacks on the nasopharynx. Parents of defective children tend to react with feelings of guilt and personal inadequacy. These feelings must be discussed and resolved if we are to secure for the child an atmosphere free from tension and overcompensation. Assistance should be given in understanding the limitations of a hearing aid and the exacting visual discrimination necessary for the child in speech reading.

Specific reading sources are the *Volta Review,* "Hearing and Deafness" by Hallowell Davis, "Developmental Diagnosis" by Gesell and Armatruda, "Opportunity and the Deaf Child" by Ewing and Ewing, and *Hearing News,* published by the American Hearing Society. If the child is in the two to three-year age group and has had no constructive guidance, his parents may enroll in the John Tracy Correspondence Course. A child who has had this instruction, when he comes to me, is a year ahead of the same aged child who has not been so instructed. The parents of our children are given detailed advice in the development of the "conditioned auditory response," and the parents themselves then become participants in a constructive program. With a play approach as a background, the child is taught to take his turn in some simple game, in response to a sound stimulus. This is the basis for ensuing audiometric and speech evaluation.

Group association with other parents of deaf children has proven helpful. At the Winthrop Foundation the parents spontaneously organized a group which meets four to six times a year and sponsors educational programs and social activities. Once a year the Winthrop Foundation organizes a group conference designed specifically for the public school teachers of our handicapped children. Here an attempt is made to include those teachers as an integral part of the remedial program and as an essential unit of our clinical research study.

Whenever possible the mother is urged to be present for her child's lessons in acoustic training, speech reading and speech development. In no other way can she develop such a keen appreciation of this formidable problem, and with expert guidance the mother gains insight and skills enabling her to continue with her child at home.

ESTIMATION OF RESIDUAL HEARING.

1. History.
2. Spontaneous Response (blinking, jumping, turning, locating).
3. Learned or Conditioned Response (gross sounds, voice, audiometer).
4. Observation of Child's Basic Language Ability.
5. Psychometric Evaluation.
6. Vestibular Function Tests.
7. Galvanic Skin Resistance Method.

The subject of hearing tests habitually calls to mind the desire for an audiogram. We are concerned here with the child's potential capacity both to hear speech and manipulate language. It is obvious that the information derived from an audiogram is limited. It tells us, at best, his threshold of awareness for several isolated frequencies of the sound spectrum. It tells us little as to his auditory performance above threshold and nothing of his discriminatory capacity upon which the hearing of speech depends. We then call upon a combination of resources not commonly used in the testing of adults.

First is the history. I have learned not to underestimate the information which may be obtained from the parents. "What do you as the mother think your child hears? (We disregard the handclap, airplane, etc.) What evidence can you recall that he has heard voice sounds? Will he attempt to repeat simple words spoken close to his ear? Will he turn to his name in a loud or moderate voice at 20 feet? Does he have any understanding of single words or phrases through hearing alone?"

Second, What have been his attempts at vocalization? Does he have the urge to talk? Does he attempt simple words to express his desires? Does he have the nucleus of a vocabulary learned through hearing alone?

The child's reflex response to sound stimuli is inconsistent, uncertain and always well above threshold. The infant under a year will blink; between one and two years he will add to his response, muscular jerks; shortly thereafter his head will turn in an attempt to locate the source of the sounds. To me, his spontaneous response has limited value, but it prepares the way for his development of a "conditioned auditory response." I use a percussion instrument, a cowbell, a police whistle and a set of pitchpipes whose range is from about 200 to 1,800 cycles per second.

The most single reliable method has been the learned or conditioned auditory response. While sitting at a small table, the child is taught to take his turn at some simple game (push a penny into the circle, a marble in the milk bottle, or put a brightly colored car into the toy garage) and he is taught to take his turn only at the command of a sound. At the first visit this can be demonstrated to the parents, who can then perfect the conditioning at home. With this method he is then tested with gross sounds as mentioned. From there we proceed to the use of voice sounds, first the vowels (either nonsense syllables or words). We are especially interested in knowing whether he can hear the consonants and he is tested with the plosives, the voiced and unvoiced consonants, and the diphthongs.

If he hears consonants with the unaided ear, he is no more than partially deaf. If he hears only an occasional vowel, loud and close to his ear, he is profoundly deaf. The next step is to try the earphones of the audiometer. I use first the frequency of 500 c.p.s. at high intensity to condition him to the technique. He is next tested at 2,000 c.p.s. since we are particularly interested in his hearing at this frequency. The majority of these children have descending curves (as related to decibels rather than sound pressure level) and if there is hearing at 2,000 c.p.s there will probably be hearing at 1,000 c.p.s. and 500 c.p.s. An elaboration of this method is the "peep show" technique advocated by Dix and Hallpike,[3] wherein there is a greater reward factor for a correct response. The child, using earphones, looks into a box of colored pictures. In order to change the picture he must pull the treadle while the pure tone stimulus is on. In this way his threshold may be approached for the various frequencies.

During the entire procedure we are observing the child's response to speech sounds, his ability to imitate, his desire to express himself.

The psychometric evaluation is desirable when facilities are available. The test is performed on a nonlanguage basis, and the more superior the ability of the child the more optimistic we may be regarding his eventual rehabilitation.

I have been impressed with the rather meager amount of additional information derived from vestibular tests. Irrigation of the child's ears with ice water effectively shatters his confidence in the doctor, who then may have difficulty in regaining his cooperation. By and large, if one is unable to elicit a vestibular reaction, the ear is profoundly deaf. If there is a significant response of the static labyrinth, he may be only partially deaf.

The galvanic-skin resistance method, as reported by Bordley, Hardy and Richter,[4] depends upon conditioning the child to a shock stimulus preceded by a pure tone. After he has been conditioned his sweat glands will respond to the pure tone alone, without the shock. This measurable response assumes a "normally reacting sympathetic nervous system." In short, it is an ingenious but time consuming method, requiring a team of trained personnel, and as yet it is not adaptable to mass testing.

REMEDIAL MEASURES.

Specific remedial measures embrace a combined program of acoustic training, speech-reading and language development. If the child has a residuum of hearing amenable to re-education and his psychometric evaluation is favorable, he is given a hearing aid. He is not "fitted" to an aid since he lacks the capacity to judge the sensations of comfort, loudness, clarity and quality. The factors considered in the selection of an aid are its cost, its past performance and its amplification characteristics as published by the American Council of Physical Medicine. The congeniality of the local hearing aid representative is also worthy of consideration. If the patient is partially or profoundly deaf, the better ear is used since binaural hearing is seldom possible. Our experience with the more powerful group hearing aids, such as the Maico and Warren Training Units has been limited.

Fig. 2 shows the remedial needs of the child in relation to his hearing capacity as a function of the audiogram. The partially deaf child should invariably have a hearing aid and lip reading regardless of whether he is able to obtain acoustic training and speech lessons. His proficiency in speech reading must not be allowed to develop to such a degree as to subordinate the utilization of his residual hearing. Recommendations for a hearing aid for the profoundly deaf child, however, should be made with caution, particularly if its procurement cannot be followed by expert guidance in speech therapy and auditory training. The profoundly deaf child needs all the speech reading proficiency he can acquire as his residual hearing can be exploited to a lesser degree.

We have at the Winthrop Foundation three well trained teachers of the deaf whom we employ on a half time basis in the hospital. The child is accompanied by his mother and has one, two or three hourly lessons weekly. Hearing is used inso-

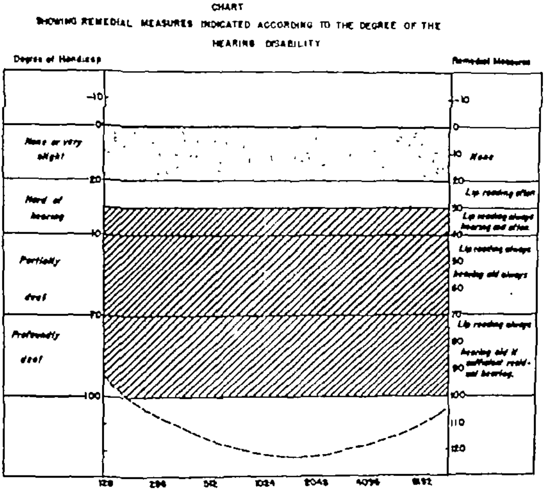

FIG. 2. Shows the remedial needs of the preschool deaf child in relation to his hearing as a function of the audiogram. The partially and profoundly deaf child also needs instruction in speech development.

far as possible, supplemented by the tactile, visual and kinesthestic senses. Since the teaching aspect of this program has not been subsidized, parents must pay for the instruction periods. In this respect it is significant that the State Department of Special Education offers no help, financial or educational, to the child in the preschool age group. We have been assisted in help with speech training and lip reading by both the Sarah Fuller Foundation and the Boston Guild for the Hard of Hearing. The Boston Guild has gradually lowered the limit for the children assisted to the five to six-year level, thus approximating the preschool age group. They offer group lessons once weekly. The Sarah Fuller Foundation, by sending its teachers directly to the home once weekly, offers valuable assistance without charge but, like the Guild, needs the services of a medical and diagnostic center where recommendations can be made. In Massachusetts we have had the cooperation of such strategically located centers as the Worcester and Springfield Leagues for the Hard of Hearing, both of which are now able to assist the preschool child in his early speech-language development. Our program has tried to integrate and utilize all of the facilities at our command.

ADEQUATE SOCIAL SERVICE FOLLOW-UP

	Known 160	Unknown 157	Total 317
Hearing Aids Recommended	73	53	126 (39%)
Hearing Aids Obtained	57 (79%)	36 (66%)	93 (74%)
Speech Training Recommended	90	78	168 (58%)
Speech Training Received	65 (72%)	46 (56%)	111 (65%)

Fig. 3. Shows the percentages of recommendations carried out in two groups of children, one known and one unknown to social service.

Fig. 3 shows the importance of an adequate social service follow-up program. Seventy-four per cent of the hearing aids recommended were purchased and 65 per cent of the patients, for whom speech training was recommended, obtained it. The percentages on all recommendations carried out were higher by 13 to 16 per cent in the group known to social service. The fact that more than one-third of the children did not receive the recommended speech training reflects the acute shortage of available trained teachers for the deaf.

Now, you ask, what results have you had with this program . . . and by what criteria do you measure success? The ultimate criterion is that of school placement. Fig. 4 shows the results of a group of 55 children, trained by us, and followed for a sufficient length of time so that their fate, as regards school placement, is reasonably certain. Of 38 partially deaf children, whose average loss was close to 70 db, 35 are in public or private schools. Only three are in a school for the deaf. Of 17 profoundly deaf children, eight are in public school.

GROUP HAVING TRAINING AT HOSPITAL

	Partially Deaf 38	Profoundly Deaf 17
Public School	29 (76.3%)	7 (41.2%)
Private School	6 (15.7%)	1 (5.6%)
Deaf School	3 (8%)	9 (52.9%)

Fig. 4. School placement of a group of 56 children who had intensive, long term speech therapy under medical supervision.

Fig. 5 shows the school placement of another group, advised and studied by the Winthrop Foundation, but receiving sporadic and somewhat inadequate speech-hearing training elsewhere. Only 49 per cent of the partially deaf and 1 per cent of the profoundly deaf children in this group were able to receive public school instruction.

Inadequate Teaching

School	Partially Deaf (59)	Profoundly Deaf (75)
Public	29 (49.1%)	1 (1.3%)
Private	16 (27.1%)	1 (1.3%)
Deaf	14 (23.8%)	73 (97.4%)

Fig. 5. School placement of a group of 134 children whose instruction in speech development was inadequate.

SUMMARY.

Facilities for the rehabilitation of the preschool deaf child in this country are inadequate. The majority of partially deaf and many of the profoundly deaf children can be rehabilitated, through early intensive remedial measures, to the level of public school placement. The enthusiasm, prestige and knowledge of the otologist are essential for the development of programs to meet the needs of these crippled children.

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ABERRANT SALIVARY GLAND TISSUE AT THE BASE OF THE TONGUE.*

COL. A. J. VADALA, M.C., U. S. Army, and
LT. COL. KENNETH SOMERS, M.C., U. S. Army,
Denver, Colo.

In a recent article, O'Neil[1] has reviewed all the cases reported in the literature on aberrant salivary gland tissue found in the tonsillar fossa region. Considering the thousands of tonsillectomies performed each year and the proximity of the salivary glands to the tonsillar area, it is surprising that only seven cases of aberrant salivary gland tissue have been reported in or about this region.

We have found infrequent references to aberrant salivary gland tissue in such other locations as the lip, palate, gums and in the vicinity of the pituitary. We have found no reference to aberrant salivary gland tissue associated with other developmental abnormalities such as were present in the case we are reporting.

REPORT OF CASE.

This seven-year-old child was first seen at the ENT clinic on Jan. 20, 1949. The main complaint was congenital microtia of the right ear. He had only a very limited vocabulary and a pronounced speech defect which his father attributed to imperfect hearing. The child had three older siblings who were normal both mentally and physically, and this was cited by the father as evidence that the child's backwardness was wholly the result of imperfect hearing.

A further complaint was that the boy was a continuous mouth breather. He had had a tonsillectomy and adenoidectomy at four years of age. The physician had told the father at the time of the operation that he was

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*From Eye, Ear, Nose and Throat Service, Fitzsimmons General Hospital, Denver, Colo.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 9, 1950.

unable to find any tonsil in the right fossa and that the condition was, therefore, a congenital deficiency. The father had deducted from this conversation that the doctor may have also missed the adenoid and that this could be the cause of the mouth breathing.

General physical examination demonstrated a well proportioned child of good nutrition. He was able to hear conversational voice but usually declined to answer questions. When he spoke it was usually monosyllabic and there was a definite speech defect. He seemed shy and was essentially a mute. He appeared to be slightly substandard mentally.

There was a congenital microtia of the right ear, there being no meatus and only a remnant of a lobule as shown in Fig. 1. The left ear was undersize and somewhat deformed as shown in Fig. 2. The external auditory canal on this side was partially stenosed. The audiogram for the left side was only slightly lowered for air and bone conduction and for the right side there was no air conduction and only questionable bone conduction. Mastoid Roentgenograms showed diploic cells bilaterally, but there was less development of the mastoid process on the right. It was the opinion of the Roentgenologist that the right internal ear and middle ear were at least partially developed.

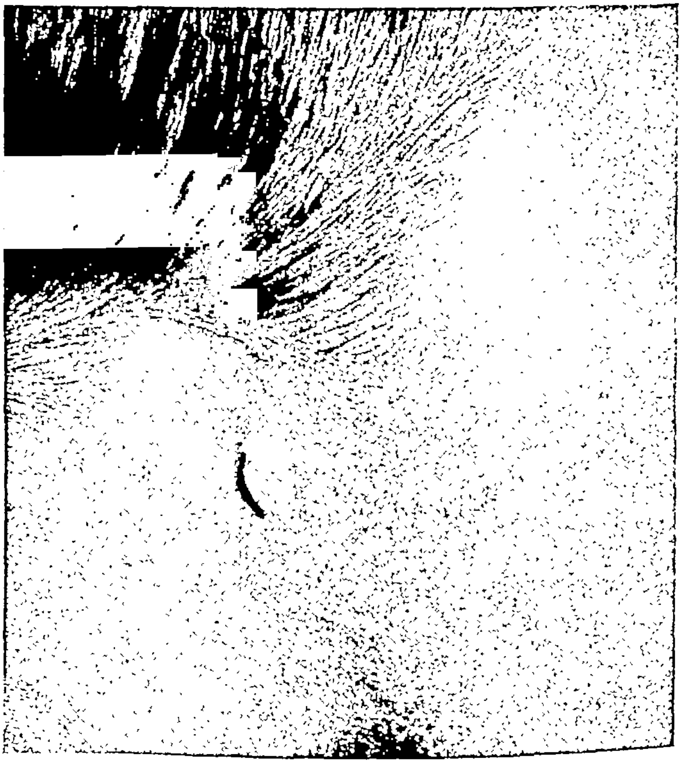

Fig. 1. Microtia of right ear.

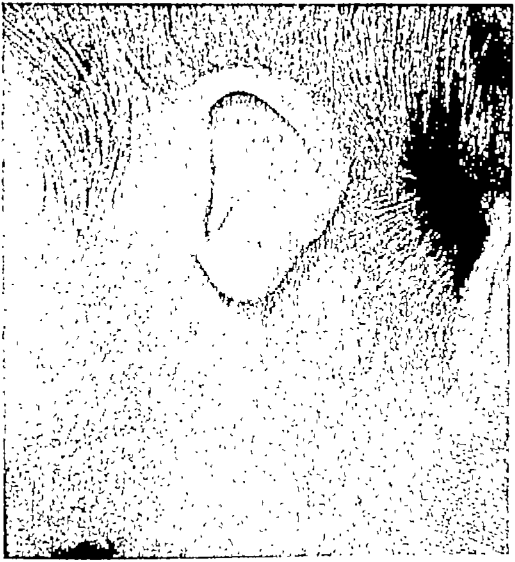

Fig. 2. Maldevelopment of left pinna.

Oral examination showed a congenital absence of the right faucial pillars and maldevelopment on the same side of the soft palate. The patient was able to blow up a balloon, to blow out his cheeks and to swallow without trouble, indicating that the muscles of the tongue, palate and pharynx were competent. The palate was highly arched, of the cathedral vault type. There was a moderate amount of pharyngeal lymphoid tissue palpated in the adenoid tonsil area. On attempting indirect laryngoscopy, a mass of tissue about the size of an unshelled almond was seen attached to the base of the tongue and below the right tonsillar fossa. At the time this was presumed to be an aberrant tonsil. This seemed most likely in view of the fact that no tonsil was present either at the time of the examination or reportedly at the time of the original tonsillectomy and adenoidectomy.

Roentgenograms of the chest showed the heart to be within normal limits of size and shape and the lungs clear. There was a synostosis between the anterior portions of the first and second ribs on the left.

The routine laboratory findings were within normal limits.

Because of the apparent difficulty with breathing and because of the speech defect it was decided that removal of the tissue at the base of the tongue might be beneficial. The operation was performed on March 2, 1949, with a tonsil snare. The tissue cut similar to fibrous tissue, and there was scarcely any bleeding, a condition identical to that reported by Hitschler and Cope.[2] The tissue was rather pale in appearance and of about the same firmness as that of a salivary gland.

The pathological report follows: "*Gross:* There is a 2.5 x 1 cm. mass covered by heavy verucous mucous membrane except for a small portion which is rough and fibrous. *Microscopic:* (See Fig. 3.) A section of stratified squamous epithelium underlaid by mixed type salivary gland tissue and one large duct. No lymphoid tissue is visible. There is considerable fibromuscular tissue around this mass."

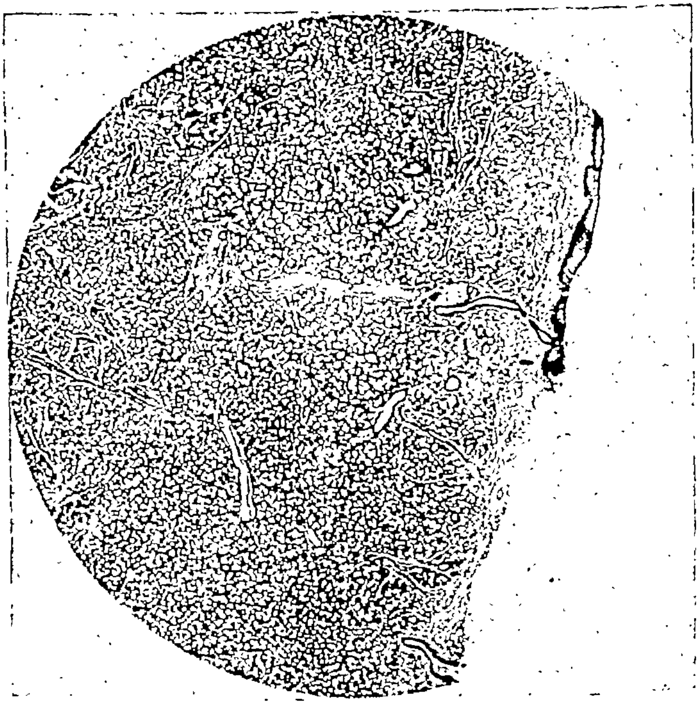

Fig. 3. Twenty times magnified histological section of salivary gland tissue.

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REACTIVE SYMPTOMS AFTER THE INTRATHECAL ADMINISTRATION OF PENICILLIN. (MENINGEAL PSEUDOEXACERBATIONS.)*

MEREI LASZLO, M.D.,
Budapest.

It is a common observation that patients who are convalescing from meningitis following penicillin therapy may suffer from headache, nausea, vomiting and fever. We formerly felt that in such cases the symptoms were due to recurrence of the meningitis, but we now realize that these phenomena are caused by the intrathecal administration of penicillin. The syndrome has been named "meningeal pseudoexacerbation." Our experiments were designed to determine: *1.* the factors which might be responsible for eliciting the syndrome during intrathecal penicillin therapy, and *2.* to find a method for differentiating between true and pseudoexacerbations.

ETIOLOGICAL FACTORS. AND SYMPTOMS.

Any of the following factors must be considered as a cause of the symptoms: *1.* spinal puncture and withdrawal of CSF, *2.* insufflation of air during spinal puncture, *3.* secondary infection, *4.* the sodium chloride in which the penicillin is in solution, and *5.* penicillin.

The untoward effects of lumbar puncture are commonly known: injury to cauda, local pain, paresthesia and neuralgia in the legs may occur and last for several days. If CSF is withdrawn, the disturbances of hydrostatic equilibrium may be associated with prostration, nausea, vomiting, headache, backpain (Birkholz[1]), and leucocytosis (Porta and Foat, and

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*From St. John Hospital of Budapest City.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 1, 1948.

Roizin[2,3]). The complaints are accompanied by no sytochemical changes. It is well known that the insufflation of air into the spinal canal may be followed by headache, nausea, vomiting and fever.

The needle employed in the puncture is passed through the skin, which is difficult to sterilize, and if serial punctures are performed the likelihood of a secondary infection is increased. Secondary infections attending lumbar puncture have been reported by Mallaret and Reilly,[4] and Smith, Duthie and Cairns.[5] The latter authors found bacteria different from those previously present in the CSF in 11 cases. The seconary infections were unaccompanied by a cellular reaction, and their course was benign. Infections with B. pyocyaneus are particularly dangerous (Botterel and Wagner[6]). Penicillin in a closed vial may be contaminated and is more apt to be so if the vial has been opened.

The possibility of the physiological sodium chloride causing untoward symptoms has not been described, and it was my desire to clarify this problem. Patients suffering from schizophrenia or epilepsy, who had a normal CSF and no fever for a week, were chosen for study. Absolute surgical asepsis was observed. The injected fluid had the temperature of 37° C. Various quantities, 1 cc., 5 cc. and 10cc., were injected into the spinal canal and cisterna, care being taken to keep air out of the syringe. We looked for any difference in the reaction between the intralumbar and intracisternal routes of the administration. The CSF originating from the cisterna was examined separately from that obtained from the spinal canal. Ten patients were given normal saline in the spinal canal and eight received it intracisternally.

Fever was one symptom encountered. The peak of 38° C. occurred five to eight hours following injection. It gradually receded but low grade pyrexia was still evident on the second day. Nine patients complained of headache and three of nausea and vomiting. Increased pressure of the CSF and pleocytosis were invariably present, and in seven cases protein content was increased. Ninety per cent of the cells were leucocytes. An increase in quantity of the saline injected magnified the CSF changes. When equal quantities were injected intracisternally and in the lumbar region greater changes were noted in those cases in which intracisternal administration was carried out.

Rammelkanp[7] and Keifer, Johnson and Walker,[8] Rosenberg and Arling,[9] Cairns, Lewin, Deuthie and Smith[10] contend that the reactive symptoms following intrathecal penicillin administration are due to high concentration of the drug, while Cairns[11] and Forray[12] attribute the symptoms to chemical contamination. We endeavor to demonstrate the conditions under which these symptoms occurred in normal individuals and the time of occurrence in patients with meningitis.

Twenty-three individuals with normal CSF received various quantities of penicillin intralumbarly[13] and intracisternally.[10]

The temperature rose to 38° C., lasted for some hours and did not become normal before the second day. Three patients complained of vomiting and 15 of headache. The pressure and cell count of the CSF was increased. The protein content was elevated in 11 cases and 90 per cent of the cells were leucocytes. The sugar concentration was determined in four cases and found to be diminished in two; in comparison with cell count the protein content was relatively low.

In four patients examination of the CSF was again carried out 72 hours after the administration of penicllin. Cell counts were six, 17, 28 and 124. Twenty to 30 per cent were leucocytes. Protein concentration was normal, and in a few cases a subnormal temperature persisted for 48 to 72 hours. These data have not been taken into consideration in the table. The penicillin was given in 5,000 to 30,000 unit doses and the reactive symptoms were independent of quantity administered. Table I shows the correlation between the concentration of the solution and the symptoms.

The reactive symptoms are milder if the concentration of penicillin is higher.

TABLE I.

CORRELATION OF REACTION AND CONCENTRATION OF PENICILLIN.

Concentration: Penicillin in 1 cc. of NaCl	Number and Percentage of Cases	Cell Count in the C.S.F.		Temperature /°C./		
		Under 400	Over 400	Normal	37-38	Over 38
1,000 units	13 100%	4 30.7	9 69.3	4 30.7	5 38.6	4 30.7
10,000 units	10 100%	6+ 60	3 30	4 40	2 20	4 40

+ = not to be evaluated: 1/10%/.

In three instances 20,000 units of penicillin dissolved in 2 cc. of CSF removed from cisterna were injected. Two of these patients were asymptomatic, whereas the third complained of fever, headache and vomiting and exhibited serious CSF changes. We assumed that this preparation was chemically contaminated.

In Table II the symptoms following the intralumbar and intracisternal injection of sodium chloride and penicillin solution are summarized.

The symptoms of pyrexia, headache, nausea, vomiting, dizziness and prostration were more conspicuous following intracisternal injection. Pleocytosis was also more marked than after intralumbar administration.

Comparison of the Lumbar and Cisternal CSF Taken Simultaneously.

This examination was performed with 17 patients, eight of whom received intralumbar, and nine intracisternal penicillin. In all cases save one the cell count and protein count of the spinal fluid were higher than those of the cisternal fluid. On the average the spinal cell count was 2.5 times and the protein content was 5.5 times the cisternal value. Three patients had a normal protein content in the cisternal fluid while that of the spinal fluid was increased.

In correlating cell count and protein content it was noted that in 11 out of 31 cases (35.4 per cent) the spinal fluid

TABLE II. DEPENDENCE OF REACTION ON THE ROUTE OF ADMINISTRATION.

ute	Number and Percentage of Cases	General symptoms:							C.S.F.					
		No Complaint	Headache	Vomiting	Temperature /°C./				Cell Count			Protein		
					Normal	37-38	Over 38	+	Under 400	Over 400	++	Under 60 mg. %	Over 60 mg. %	+++
bar	23	13	8	2	8	7	5	3	12	10	1	14	6	3
	100%	56.7	34.7	8.7	34.7	30.4	21.7	13.2	52.1	43.4	4.5	60.8	26	13.2
ernal	18	3	11	4	2	3	13		7	11		12	6	
	100%	16.6	61.2	22.2	11.1	16.6	72.2		38.8	61.2		66.6	33.4	

= not to be evaluated.

exhibited a normal protein content and high cell count. The same correlation was found in the cisternal CSF in 13 out of 25 instances (58.3 per cent).

REACTIVE SYMPTOMS IN MENINGITIS.

During the period 1946-1948, 31 meningitis patients in our hospital were treated with penicillin. In the most severe stage of the disease intrathecal penicillin therapy was not attended by any reaction; reactive symptoms did not appear until the recovery process had started. Pseudoexacerbations were observed in eight cases; in six of them the symptoms occurred when the spinal CSF cell count ranged from 30 to 160 and the protein count from 35 to 65 mg. per cent. In the seventh patient the cell count was 320 and the protein content 75. In the eighth case the reaction began when the spinal fluid cell count was 1,600 and the protein content 45 to 60, while the cisternal fluid cell count was 200 and the protein content 30. Reactive symptoms coincide with cell counts of 100 to 200 and a protein content of less than 45 to 60 mg. when the recovery from meningitis has started.

Examinations to Differentiate Between True and Pseudoexacerbations.

In 1946, our experience with penicillin therapy in meningitis was limited and we were unaware of the reactive symptoms due to its use. For this reason, in our first cases we were puzzled when the meningeal pseudoexacerbations occurred. At first we thought that the meningitis had become worse and continued to administer penicillin for a time while the symptoms became more marked.

Case 1: A boy, age 13, was admitted Sept. 29, 1946. Six days prior to admission he developed pain in right ear and spontaneous discharge on the following day. The temperature was 39° C. Everything he ate was vomited, and he lay motionless. He had lost consciousness on the morning of admission and had fully developed meningeal symptoms and purulent spinal fluid; 20,000 units of penicillin were given immediately. The left ear was intact. On the right side there was profuse discharge and the drum was swollen and hyperemic. Radical mastoidectomy was performed with wide exposure of the dura of the middle and posterior fossa. There was extensive pneumatization reaching to the zygomatic root and posterior to the lateral sinus. The cells were full of pus, and the dura of the middle fossa was markedly hyperemic. Transfusion, cardiac stimulants and penicillin were administered.

Return to consciousness and general improvement were noted on the first postoperative day. That evening high fever, delirium, headache, nausea, nuchal rigidity and positive Kernig's sign appeared and the CSF pointed to deterioration. Recovery was rapid when intrathecal penicillin injection was halted.

Case 2: A 25-year-old woman was admitted on Nov. 14, 1946. She had been treated for pulmonary tuberculosis with pneumothorax for two years. For four months prior to admission she had suffered from purulent tuberculous otitis media. For three days the fever had ranged from 38 to 40° C. and auricular and retroauricular pain, chills, fever, nausea, vomiting and headache were present.

State on admission: There was neck rigidity. Kernig's and Brudzinski's signs were absent. The skin was hyperesthetic, the tongue and pharynx dry and furred, the reflexes spastic. Ophthalmoscopy revealed dilated veins, sharply limited papillae and normal pupillary reaction on both sides (Dr. Mócsy). Spinal fluid was clear and under increased pressure. The right eardrum was intact. On the left side a purulent discharge was found in the external meatus, the drum was swollen and hyperemic. Landmarks were absent. There was an anterior inferior perforation with pulsating discharge. The mastoid and jugular vein areas were tender to pressure. On Nov. 15 antrotomy with exposure of the lateral sinus was done. The cells were medium sized and near the apex they were lined by intact mucosa, whereas at the antral region the mucosa was swollen and the cells were filled with serohemorrhagic and seropurulent secretion from which streptococci were cultivated. The anterior sinus wall was grayish and thickened, and the lumen contained a red thrombus which was removed by incision and suction. Penicillin was given, first intramuscularly three times daily (20,000 units per dose). After CSF examination intrathecal administration was begun. On the first postoperative day the neck was mobile, and the general condition much improved; but later nausea, vomiting, prostration and rigidity of the neck appeared. The spinal fluid was repeatedly hemorrhagic, so occipital puncture was performed, and penicillin was injected into the cisterna (20,000 units in 20 cc. of sodium chloride). Prostration, vomiting and cervical rigidity became more marked. Quick recovery followed cessation of penicillin therapy.

In this case the intrathecal administration was thoroughly superfluous, the CSF being normal; however, the laboratory reports had not been received before the second day. Irritative symptoms suggested the presence of an intracranial complication, hence we were reluctant to discontinue intrathecal administration. Finally, recovery immediately followed its omission.

It was these cases that induced us to perform experiments with intrathecal penicillin administration in so-called normal cases. As may be seen, the reactive symptoms ceased in 48 to 72 hours. For this reason the problem can best be solved by observing the pseudoexacerbation after the omission of penicillin. This procedure would be dangerous in some patients, so efforts were made to find a method by which true recurrences could safely be differentiated from pseudoexacerbations.

It was stated that, after the intrathecal administration of penicillin, the cell count of the spinal fluid was 2.5 times its protein content, 1.5 times as high as the values found in the cisternal fluid. We further found that the protein content of the spinal fluid was pathologically increased. In some instances this was proven by normal protein content of the cisternal CSF. In an effort to apply these phenomena clinically, we performal simultaneous tappings in dubious cases. Results of simultaneous punctures cannot be evaluated in meningitis unless the findings are compared with the results of previous punctures.

The following cases exemplify the differentiation between recurrence and pseudoexacerbation by comparing the findings of the spinal and cisternal CSF taken simultaneously.

Case 3: A man of 46 was admitted June 12, 1947. Symptoms of ear pain; fever, retroauricular pain and no discharge were experienced three weeks before entry. Later the pain ceased but the hearing remained impaired. Five days prior to admission, fever, ear pain and retroauricular pain recurred. Headache, chills, fever and vomiting followed two days later. He was comatose on admission. Sulfathiazole was given. On the following day the patient regained consciousness while meningeal symptoms persisted. The right tympanic membrane was swollen and hyperemic; landmarks were obliterated. The mastoid region was tender. Intramuscular and intrathecal penicillin were introduced. Antrotomy was done on June 14. Pneumatization was microcellular, and the mucosa was hyperemic and edematous. The periantral and perizygomatic cells contained much pus.

Early omission of intrathecal therapy resulted in a true recrudescence as seen from the findings of the CSF taken on June 20. The cell content of the cisternal fraction was higher than that of the spinal CSF and the protein concentration of both fractions was increased. Sugar content was diminished. Ninety-eight per cent of the cells were polymorphonuclear leucocytes. Bacteria were demonstrated in the CSF. On June 23, CSF findings showed undoubted improvement of the meningeal process: the cell count of the spinal fraction exceeds many times that of the cisternal CSF while the protein content of the cisternal fluid is nearly normal. The sugar level was higher in the cisternal than in the spinal fraction, which is the normal state. The percentage of lymphocytes gradually increased. We then injected a last dose of penicillin (10,000 units) into the cisterna. The following day penicillin was stopped in spite of the increasing cell count. The patient recovered.

The problem was still more difficult in the next case. Though the simultaneous punctures pointed to a pseudoexacerbation, we continued the intrathecal therapy because we did not hold the symptoms reliable.

Case 4: A man, aged 24, was admitted Dec. 5, 1947, with the history of recurrently discharging ears since the age of three years. The hearing had been failing progressively. He had two attacks of meningitis, one in 1926 and one in 1944. Two weeks before admission he developed an acute cold. Both ears became full and tenderness appeared in the retroauricular region. Relief followed in three days and he was apparently well. On the day of admission, chills, fever, headache and vomiting occurred.

State on admission: Meningeal symptoms, clear sensorium. Thick fluid in the right external ear canal. Tympanic membrane swollen and red, with obliteration of anatomical detail. No mastoid tenderness was present. Intramuscular and intrathecal penicillin were given.

On Dec. 7, intralumbar and intrathecal penicillin injections were given. The patient then complained of malaise and headache, and vomiting ensued. The cell count of the cisternal fraction was 200 as compared with 1,600 in the spinal fraction and the protein concentration was nearly normal. The high 9,600 cell count obtained the following day was undoubtedly due to the intrathecal administration of penicillin. Being ignorant of the diagnostic value of the comparative examination of the spinal and cisternal CSF, we continued the intrathecal penicillin until a cell count of 30 was obtained.

Case 5: A 36-year-old man was admitted Dec. 6, 1947. His complaints of thoracic pain, cough, fever and headache have been evident for one week.

State on admission: Periodic delirium, meningeal symptoms, bilateral pneumonia, jaundice. No otolaryngologic abnormality was noted. Intramuscular and intrathecal penicillin therapy was introduced.

Following penicillin therapy the fever receded, and the CSF exhibited improvement. On the third day the fever returned, and nausea and headache were experienced. On Dec. 10, simultaneous punctures were done. The CSF showed the signs of uneventful healing, but the general symptoms became worse. Believing the symptoms to be due to intrathecal penicillin, this route was abandoned while intramuscular doses were raised. Sulfadiazine was started. CSF findings on Dec. 12 corresponded to the reactive symptoms elicited by intrathecal penicillin therapy. This assumption was confirmed by the fact that the patient felt well and was afebrile on Dec. 13. On Dec. 14, he again developed hyperpyrexia. Recurrence was assumed though meningeal symptoms were absent. For this reason simultaneous punctures were performed on Dec. 15, and penicillin was injected into the cisterna; however, the findings indicated recovery. Thus the findings of Dec. 12 could have been due to the reaction to intrathecal penicillin.

An experiment was then performed on a 72-year-old woman who was improving following a pneumococcic meningitis. On the sixth day following the omission of intrathecal penicillin therapy we injected 10 cc. of sterile physiologic NaCl into the spinal canal. In the CSF taken before the injection the cell count was 11 and the protein content 90 mg. per cent. Twenty-four hours later the cell count was 126 in the spinal fluid and 39 in the cisternal fraction. Protein content of both samples was 75 mg. per cent.

Meningeal pseudoexacerbation was encountered in three cases. In two the presence of pseudoexacerbation was obvious, whereupon the intrathecal therapy was discontinued. In the third patient there was doubt and the diagnosis was made by means of simultaneous tappings.

CONCLUSIONS.

On the basis of the data of literature and our own experimental findings it could be stated that the reaction due to intrathecal administration of penicillin may be due to the separate or the simultaneous action of several factors. Spinal puncture and withdrawal of CSF led to general symptoms only, whereas the injection of air, sodium chloride or penicillin may give rise to changes in the CSF also. The symptoms following the intralumbar and intracisternal administration, respectively, are not quite identical. In the majority of cases intracisternal injections are followed by high fever and marked general complaints and a higher degree of pleocytosis. From the viewpoint of the evaluation of the symptoms of reaction and in order to reduce reaction to intrathecal penicillin, the following principles should be taken into consideration: aseptic technique must be observed; the vial containing penicillin should not be opened prior to the intrathecal injection; penicillin is to be dissolved in the CSF. If physiologic sodium chloride is to be applied as the solvent, the right proportion is 10,000 units per cc. When the process is subsiding the intralumbar route should be chosen without insufflation of air except in cases where adhesions are to be prevented.

In the course of a meningitis reactive symptoms to intrathecal penicillin administration do not occur. This is understandable, considering the fact that while the meningitis persists, two stimuli act at the same time: the infection and the penicillin. When the irritation due to the infection persists (dilation of vessels, emigration of cells) the administration of penicillin has no sequelae; however, when healing begins, the introduction of a new irritant is followed by reaction. Intensity of reactive symptoms also depends upon the age of the patient and the duration of the meningeal process. In the presence of old age or long standing disease the functional capacity of the tissues is restored with difficulty; therefore, the protein content of the CSF, due perhaps to the lasting damage of the vessel wall, may be high for several months after the clinical recovery.

When a penicillin reaction appears during recovery it has a prognostic value. It points to the onset of recovery. It may be inferred from this fact that the intrathecal administration of penicillin should be omitted when these reactive symptoms ensue; that is, when the cell count of the CSF does not exceed 100 to 200.

Pseudoexacerbation can best be distinguished from a true recrudescence by the temporary interruption of the intrathecal penicillin therapy. Reactive symptoms recede in 24 to 72 hours. If the interruption of therapy is considered hazardous the two conditions can be differentiated by the examination of the spinal and cisternal fractions of the CSF taken simultaneously. True recurrence may be assumed if the cell count of the cisternal fluid is many times that of the spinal sample, the protein concentration increased in both fractions and a predominance of polymorphonuclear leucocytes (over 90 per cent). The pathogenic microorganisms do as a rule reappear; however, their absence is not conclusive evidence against a true recrudescence. Pseudoexacerbation on the other hand is characterized by a higher cell count in the spinal fraction than in the cisternal sample and a closely normal protein content in both; if, however, there is an increase in proteins it occurs in the lumbar fraction. The sugar level is normal or slightly reduced, and its value is somewhat higher in the cisternal sample. Though polymorphonuclear leucocytes may be increased, there are fewer than 90 per cent. If bacteria are present they are saprophytes.

SUMMARY.

1. The intrathecal administration of sodium chloride is followed by symptoms of meningeal irritation. Similar effect is exerted by penicillin.

2. The symptoms are independent of the quantity of penicillin, but they are milder if the drug is injected in a more concentrated solution.

3. Penicillin should be dissolved in the CSF or if sodium chloride is employed as the dissolvent, the right proportion is 10,000 units per cc.

4. The intrathecal (lumbar or cisternal) administration of penicillin is followed by a change in the cell count and protein content. The cell count and protein content of the spinal fluid is 2.5 times and 1.5 times, respectively, higher than the corresponding values of the cisternal fluid. The dissociation of the cell count and protein content is less marked in the spinal fluid than in the cisternal one.

5. Intralumbar injections are usually followed by less intensive symptoms than are intracisternal injections.

6. Intrathecal administration of penicillin has no sequelae in meningitis. A reaction during recovery indicates that the process of healing has started. At this time (cell count 100 to 200) intrathecal penicillin therapy is useless or harmful.

7. Recurrences and pseudoexacerbations may be differentiated by temporarily interrupting intrathecal penicillin therapy or by simultaneously examining cisternal and lumbar fractions of CSF. The cell count, protein level and sugar content serve as guides in the differential diagnosis.

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MEDICAL ILLUSTRATORS' DIRECTORY AVAILABLE.

The Directory issue of *Graphics*, the official publication of the Association of Medical Illustrators, contains the name, address, training, professional experience and reference to major published work of each member. Other information pertaining to the profession is included.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9.

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National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

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Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

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The dress that needn't have been so beautiful

THERE'S NO REAL REASON for a girl to have the most beautiful dress in the world. Even my daughter Sally.

But—I bought it. And when I paid the bill, I whistled! Partly with the well-known father's bill-shock. Partly for happiness. Because, Sally was right—there never *was* a prettier dress to get married in.

It's times like that—when we can buy something really important even if it *is* a luxury—that I feel like such a lucky guy.

I know the luckiest day of my life was when I signed up to save regularly through the Payroll Savings Plan at the office!

Buying U. S. Savings Bonds . . . by the Payroll Savings Plan or the Bond-A-Month Plan . . . is the world's "foolproofest" method of saving money. And every $3 you invest turns into $4.

Automatic saving is sure saving—
U.S. Savings Bonds

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THE

LARYNGOSCOPE.

VOL. LX JULY, 1950. No. 7

MEDICAL EDUCATION.*

(Presidential Address.)

ROBERT C. MARTIN, M.D.,
San Francisco, Calif.

The title of this address is misleading in that I have decided to omit a formal discussion of curricula and costs. Instead, I propose to challenge the idea that "all is lost" and that a young man should not consider medicine as a career. You must continue to try to get the best type of young men and women to enter our profession and you will get them only if you abandon the defeatist attitude which so many older men now have. We want the type of man who accepts responsibility and is not interested primarily in trying for an easy life and regular hours. The latter type is literally a "dime a dozen" and will add nothing to medicine.

What follows is my personal belief and advice to the many young people, including my own son, who come to me for advice. They and their parents ask whether the shadow of forthcoming events indicates that our profession may degenerate into a conflict for political preferment in which the art of politics may supplant the art of medicine, and the science of medicine deteriorate to serve political dialectics and dogma;

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*Presidential Address at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 25, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1950.

in short, whether service to the state may become the all-important thing and service to humanity and the individual purely a secondary matter.

We must not lose sight of the fact that we enjoy the highest standard of spiritual freedom, education and material living in the world. Nowhere else in the world is it so uniformly possible for everyone and anyone to get an education and rise as high as his capabilities permit. These factors are mentioned to show that it must be difficult to persuade people enjoying these privileges to substitute something as a system which promises far more but delivers little. It would be trite at this point to cite certain well known schemes in other countries, which seem very definitely to be found wanting.

You may ask, "But what of the growing spirit of radicalism in our schools? Does it not indicate a desire for social reform?" May I answer that I do not like that word *reform.* As I understand *reform,* it means a change of conditions for the better and not retrogression. I insist that to go back to stateism is regression — not progress; and reform is not the word. "Social change," if you wish, but not reform. It reminds me of the furious appellations of "reactionary" that issued from the empty brains and open mouths of so many do-gooders in late years. In reality those of us who are so tarred and feathered are the progressives and our critics who advocate regression to an all-powerful state are the reactionaries.

The spirit of radicalism is a desire for change rampant in all normal youth; but this younger group is a minority, vociferous out of all proportion to its size. It will soon become mature and disillusioned as to the value of stateism and the political machine of perpetual motion where no energy is introduced, but from which much issues. They will soon learn that the philosophy of "something for nothing" in any society is no more possible than is perpetual motion in the field of physics. This desire for social change is a normal condition in youth and does not indicate that we are headed for the rocks. I believe it was Voltaire who said, "Show me a man of 25 who is not socially conscious, and I will show you one who is lacking in human sympathy; but show me a man at 45 who is still markedly so, and I will show you one utterly lacking in intelligence." This perhaps is an overstatement, as most of us who are 45 or over are socially conscious; but we hope this consciousness is leavened with experience and understanding. One cannot literally bump one's head against special privileges, be the special privilege that of a rugged individualist, bureaucrat or professional loafer, without tempering his desire for the milk of human kindness with the element of human justice; but until one has had those bumps, he has no yardstick by which to measure the materialism of the average human, as manifested unfortunately in our everyday life and to which all of us are in a measure a part. Is the spirit of youthful radicalism and advocacy of social change to be decried? No! Without it there would be little progress and it is most necessary to stir up things with thought even though one does not agree with that thinking. I believe that the counterbalance of maturity and experience is sufficient to direct and modify this energy and unrest so that the fusion of both attitudes may become a workable entity to the advantage of all of us. This progress will not be an even line but will be irregular and characterized by excesses of zealots on either side, but I believe wholeheartedly that the graph will show a gradual evolution of a system of medicine better for society and for the presently essential but politically forgotten man, The Doctor.

Most of you studied medicine because you enjoyed the relationship with humans as individuals and with an idea of helping them through pain, sickness and personal problems. With most of you it was necessary that you be self-supporting in order to do these things, and you hoped to be given the privilege of providing for your old age. I further believe that you studied medicine because you enjoy the individualistic type of life you are allowed to lead, even with its attendant responsibilities and being continually tied to the telephone. The type of medicine you entered, whether it took the form of surgery or medicine, depended upon your bent. A minority blessed with an investigative turn of mind did research; again with the idea of helping the human individual. Certainly monetary gain never led anyone into the field of research. I challenge the statement that any of you studied medicine entirely for pecuniary reasons, for if you did you showed a low order of financial intelligence. There are far greater gains in dollars and cents made in other fields without the surrender of the personal freedom that a successful medical career entails.

I do not believe that a bureaucratic system with the Utopia of financial security and regular hours for the doctor appeals to the prospective student. The former is a figment of the imagination and the latter, theoretically desirable though it may be, robs medicine of its greatest burden and its charm which is individual responsibility. For one, I do not want the next "shift" to take over a patient about whom I am worried! That is my responsibility.

My son says, "Dad, I would like to study medicine." My answer is, "Fine, if you are sure that is what you want. The pendulum may swing to lower standards and loss of individuality, but people cannot be fooled all of the time and some day it will swing back and I hope settle to a midpoint of common sense. It may be after your time before another Magna Charta can be forced from the hands of reluctant privilege holding politicians. As surely as I know human nature after years of practice, you will find that both donor of service, the doctor, and recipient of service, the patient, will eventually fight to retain or regain their individuality and self-respect.

"So go ahead, if you are certain you really want to study medicine. It is a good, though rugged way of life. You will miss taking my grandchildren to football games and to the theatre many times because of some imaginary ailment of one of your patients; but the real emergency which is often there when you arrive will give you compensation for the false alarms, and the missed good time.

"Stand for what you believe is right and don't go along with the crowd just to be popular, and you will be glad that you have studied medicine; and I think you will have a good life even though changes come, changes to which your father and older men will have difficulty adapting themselves. You will not have that difficulty. You cannot find a more satisfying way of life nor one where you can help people more. We older folk have tried to do a good job. You will doubtless do better."

MEMORIES OF POST GRADUATE TEACHING.*

HARRIS P. MOSHER, M.D.,
Marblehead, Mass.

First I thank Dr. Martin for the high honor of choosing me as the Guest of Honor of this meeting of the Society under his chairmanship. It is deeply appreciated. It brings back very pleasant memories of when he was with me as a student in the rather distant past. He was outstanding then, and, as you know, has continued that characteristic.

On looking at the members of the Association before me, it is easy to see three or four faces whose looks speak louder than words, and say, "Will he never quit and give us a chance?" The October issue of the *Saturday Evening Post* of last year carried the sentence, "The late Dr. Harris P. Mosher." This should give these men hope; but I find it a queer sensation to be both dead and alive.

The psychiatrists say that confessions are the catharsis of the soul, so I have a few to make. My first is that I shun the word "old." To speak it brings crashing down upon me the heavy weight of the accumulated years, and I find that I am becoming as sensitive about telling my age as a woman over twenty.

Second, you can date me by the following incident. At a neighboring hospital where I recently had a check-up, the nurse who prepared me for a chest X-ray and a day or two in the hospital was heard to say, "Think of it, I have just found a man who still wears garters."

Third, show me the photographs of your children once but, please, not every time we meet. Be merciful, as I have no

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*Read at the dinner given by the local and Western members to the visiting members of the American Otological Society, American Laryngological Association, the American Laryngological, Rhinological and Otological, and the American Bronchological Societies, May 27, 1950, at Fairmont Hotel, Nob Hill, San Francisco, Calif.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

children. Remember that I, too, was a beautiful golden-haired baby; at least my mother had to say so, in order to keep up with all the other mothers.

Fourth, my bluff, staccato manner is deliberately put on. When I was in flower, so to speak, people came to me because they wanted something, and this manner brought them at once to the point. I found it very useful, and survived in spite of it. From the first I have been conscious that neither Chesterfield nor his successor, Emily Post, nor Dale Carnegie, would recommend it.

So much by way of introduction. My topic tonight is "Teaching." All that I have to say is reminiscent of my post graduate courses. All that I know about teaching I learned in them. Half or more of what I have to say is in a light vein; the rest, at least to me, is serious.

My courses featured applied anatomy. I held that it was only if you knew your anatomy were you ready for surgery. The parts of my post graduate course which I hoped to be of the most lasting value were: First, a comprehensive series of drawings of picked anatomical and surgical specimens. A drawing was always started in the tracing box, and then finished according to the student's native skill in drawing, and with what help I could give him as he went along. Second: A series of casts to supplement the drawings was made from master casts or from the cadaver. The casts, unlike the drawings, were in three dimensions and constituted an anatomical and surgical library which ranked next to the cadaver for study, and was always available.

I have always liked to work with my hands. To measure a significant anatomical or surgical specimen, to draw it, or to reproduce it in permanent form was an inborn and always an increasing and lasting pleasure.

The universal remark made by the students at the beginning of a course—and it rings plainly and plaintively in my ears, even yet, was: "I cannot draw." My answer to this always was: "Trace,—use the tracing box. An accurate tracing is worth more than a pretty, inaccurate drawing. Measure everything, and record the measurements on the drawing." I have long had the feeling that repeated measurements leave a record in the cerebellum, and when a motion becomes automatic the cerebellum outranks the cerebrum in bringing this about. This is the reason why I have the students measure everything. It is the job of the cerebellum to regulate and coordinate movement. Regulating is but another word for measuring.

My post graduate courses began in 1898 and ended in 1939, roughly some forty years. They were interrupted only by the first World War.

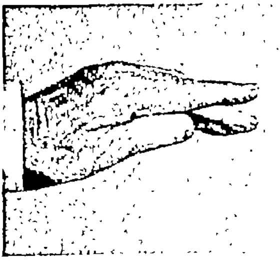
I had the good fortune to have mature men,—no G. I.'s. I found the ideal class to be ten,—with two or three incoming house officers as strikers. For a short time I had twenty men which practically made the course pay the medical school's share of my salary. I always felt that the administration considered this the most important thing about the course. In the beginning I paid for my own school equipment.

I soon found that I could not give a course without stopping private practice. Frankly, I did not have to give up much. For seven weeks I lived at the medical school. I was there every day until five o'clock, but I admit that I sometimes watched the clock. Often, I made a surprise evening visit. We all dined together at noon in Vanderbilt Hall, the medical school dormitory. At the end of each course there was a party for the men and their wives. The instructors who helped me came, too; the chief ones were Dr. Berry, Dr. Schall, Dr. Knowlton, Dr. Kazanjian and Dr. Mueller. They have been thanked before, but I take this, and probably my last, opportunity, to thank them again for their faithfulness to me, and the cause of teaching.

The course always began on a Monday night at my house at the unfashionable and rather shoddy end of Beacon Street. This was our "coming out party," and a chance to get together a bit socially. It proved very useful because I could begin to size up the men and they could do the same with me. There was generally a blizzard on this night. I told the men what I expected of them, especially their working hours, which were from nine in the morning to ten at night, Sundays and holidays. These may seem pretty long hours, but because of them I soon stumbled upon a bit of useful psychology. This was that the men when they got home bragged about their slave life with me, and I capitalized on this and never shortened their hours.

At the coming out party I announced that the wives the men brought with them were to consider themselves actual widows for the coming seven weeks. Often some of the men had already been accepted as house officers at the infirmary and were used in the course as helpers. Naturally, their wives, if they brought any, stayed on. Most of these took jobs to help out with the pathetically simple homes they were able to establish. I am writing these few lines to express my admiration for these plucky souls, even if one of them, in order to make sure of her man, married him almost on the way to the railroad station.

Each week ended with a quiz. I soon found that in order to make this worthwhile and not simply a parrot-like exercise, I had to arrange it carefully. On one of the quizzes after the men had finished their work on the ethmoidal labyrinth, I gave to each man or, rather, tossed to him, baseball fashion, a ball of clay. This purposely was made large. I gave my thumb and forefinger measurement as a rough guide and asked them to make a model of the ethmoidal labyrinth with eyes shut. This measurement on my hand is two inches and is the dead-line measurement in the nose.



On my hand, from the tip of the thumb to the tip of the forefinger, is two inches.

Half of the models which were turned in were passable, but three or four would have done for a cow or an elephant. After the exercise the class were an humble lot, and this rather pleased me.

I was proud of the student lectures. Each man was assigned one and worked over it as though his life was at stake. To illustrate their lectures they almost depleted the medical school museum. The poor devils had the habit of rehearsing in the nearby washroom, and often found relief in all its facilities.

To turn from my course for a moment to the teaching of the third year class of the medical school, — I still hold, although lectures are now somewhat in disrepute, that a limited number of lectures are worthwhile and necessary, to correlate the specialty with general medicine and surgery. If a lecturer cannot, from his experience, give a perspective of his specialty not to be found in the textbooks, he is not worth his salt as a teacher. To give this correlation to third year students should be his first and chief job.

The following are some of the little happenings at the courses:

Young Lynch, now not so young, saw snow for the first time when he came to the infirmary as house officer. He and his wife lived on the northern slope of Beacon Hill, which then, as now, was the Latin Quarter of Boston. When the first snow storm came along, he rushed out of the house to feel of it, and to play in it like a child.

Bacher, of San Francisco, saw his first snow in the courtyard of the medical school, and he began at once to eat it.

At a get-together party, I noticed that one of the men wore a large diamond ring. I never had a diamond ring and never expect to. So, I could not help saying that the dissecting room was no place for diamonds. I turned my head, and when I looked back the man had slipped the ring off his finger into his pocket, and I never saw it again.

Every three or four years the weather was mild enough for the men and their wives to come to Marblehead. On one of these occasions, we all had lunch together in neighboring Salem at the Hawthorne Hotel. Mrs. Mosher sat at one end of the table and I, correctly, sat opposite her at the other end. After the party was well underway, I looked up and could not see her. When she was fished out from under the table, it was found that the legs of her chair had collapsed· Examination showed that four or five other chairs were in condition to do the same. These were replaced, and the luncheon continued normally to the end.

There were two Belgians, each in a different year. One was asked to give his description of the course and said, "Mud, plaster and glue." The other when at Marblehead, was standing with Mrs. Mosher in the garden, looking out over the harbor, and broke the silence with the remark, "You have a fine belly view." He was right.

Another year again there was a man from overseas. It turned out that he never had had a hammer in his hand, so never had driven a nail, and knew a rake only by name. Our lawn had just been fertilized. Mrs. Mosher, out of pure deviltry, gave him a rake and asked him to help her clean up, and he was gallant enough to do it. He probably was something of an aristocrat at home; but I could not help thinking that he had missed most of a boy's real joys, that is, working with his hands. In England and on the Continent, home life ceases when, around twelve to thirteen, boys are sent to boarding school, and from then on become young gentlemen. If this is what had happened to him, I suppose he signed checks at fifteen, complained of his allowance, and at his university had a mistress.

Marblehead has some very crooked streets,—London none more so. It was my custom to bring my car full of "Course" men by what I announced to be the straightest road in Marblehead, but really was the most crooked. Usually the men were duly astonished, but once one of the men remarked that he preferred the straight roads of the west where the intersections were always at right angles, and you could go 500 miles without reaching your next-door neighbor. Naturally I had no *further use* for him.

At another time, there was a man who had never seen the sea. Just beyond our house there is a straight and clear shot for three thousand miles across the Atlantic to the Bay of Biscay. When the tide is high, the water of Marblehead Bay reaches within ten feet of the retaining wall of our garden. As soon as this man reached our house, he rushed down to the water's edge and dished up a handful of water to find out if it was really salt. It was very evident that he did not swallow it, and that one mouthful was enough.

Kennon and his running mate, both from the upper South, each had a keen dry wit which he used constantly, and kept the members of the course in a chuckle. Both had a tinge of the soft Southern voice which many of us here in the North envy. Kennon was tall and his mate short, so that they soon became known as Mutt and Jeff.

At the party on the first night of their course, one of them—I think, Jeff—produced a bottle of White Mule, and gallantly offered it first to Mrs. Mosher. It smelt and tasted like hair oil or worse, and she could not get it down. None of the rest of us could either, so the two of them quickly emptied the bottle, alternating drinks so that each got his full half.

A short time before Kennon's death about two years ago, he said in a letter, "I am hungry to see you again." I never had comradeship expressed more delicately, or deeply.

The party at the end of the course was an open season on me. I was fair game, and the men made the most of it. The instructors and the various members of the class also came in for gibes. One year just before party time, one of the men had a birthday. At the party, he was presented with a plaster of Paris birthday cake. As it was being handed to him, it was purposely dropped—it fell with a deadly, uncake-like thud, and broke to pieces. When the applause and laughter died down, Mrs. Mosher presented him with a fine, proper cake. The afternoon party over, we usually went for dinner to the Wayside Inn of Longfellow fame. At the dinner there often was a presentation. Over the years, I received an assortment of gifts varying from goldfish to a gold watch. Bacon, one of the wisest heads the English world has ever known, says in his essay on Fortune,—"And certainly there be not two more fortunate properties than to have a little of the fool, and not too much of the honest." These parties were silly, foolish affairs, but they ended the tension of the previous weeks. For those who raise their eyebrows at them, I have this to say: "I pity their wives for the empty lives they have to live with them."

Now, a word more about teaching.—It is becoming increasingly hard to get young men in our locality to give up time from their practice for teaching. All teaching should be paid for. The amount depends upon the budget of the department. In my beginning days, we were supposed to do anything we were capable of doing for the older men, for nothing. Happily, those days are over. Getting a practice is harder than it used to be, especially in a large city, and raising a family today is more costly. I feel that I have led a sheltered medical life because, thanks to my father, and later to Mrs. Mosher, I had a small private income from the start.

Speaking generally, when you try for a reason why a man should take up teaching, the first one which comes to mind is that he does it to make a living, and does it in spite of the fact that the living proves to be a modest, if not a meager one. The crudest explanation of all, one which is only a poor wise crack, is that, if married, he cannot be boss at home, but hopes he can in the school room.

When you are honest with yourself, you realize that one of your most constant dreams is to leave something behind to be remembered by, and you hope that something in your teaching may do this. One remark of mine, perhaps, may do this for me, namely: "Respect the virginity of the nasofrontal duct, or take the consequences."

I cannot help feeling that in teaching there is an inborn element of the urge to lead and direct, as in the leader of the herd or the mob; but it is hard to believe that those teachers who live in ivory towers, sung, warm and protected, like a human caterpillar in a cocoon, have this feeling for leadership.

In closing, allow me a general remark or two about teaching. In speaking, a teacher should radiate enough strength to command attention, and, if possible, respect. A weak voice and manner have no place in a class room at any grade. More than once I have said to my students that they should feel that what they were saying in their assigned talks was the most important thing being said in the world at that moment. Without being brazen, they were to put some strength into their voice and manner.

A few teachers help to make their points as much by their mannerisms as by what they say. I am not advocating clowning, but action is life and life is contagious.

As I see it, the fundamental fact about teaching is that the teacher's personality is what makes him successful or a failure. To attempt to define personality would soon put me over my head in metaphysics, and at once put you listeners to sleep. One set of thinkers hold that God has no personality; fortunately, we as humans have. Philosphers often rank personality as the basic, divine spark in a human being. There is truth in this, even if it does sound like tall talk.

One thing about teaching seems clear, at least to me, and this is that every teacher, consciously or unconsciously, has the instinct of a showman and, at his best, is a showman. I refrain from naming present-day examples.

127 Front Street.

POLIOMYELITIS-TONSILLECTOMY SURVEY — YEAR 1949.*†

DANIEL S. CUNNING, M.D.,
New York, N. Y.

The statistical report which I am about to present completes a four-year nationwide survey conducted by this Society in an effort to determine whether or not there is any relationship existing between poliomyelitis and tonsillectomy and, if so, whether it be causal or coincidental.

Ever since the greatly publicized family of five children in Akron, Ohio, developed bulbar poliomyelitis following tonsillectomy some years ago, epidemiologists, The National Foundation and State Boards of Health throughout the country have strongly advised parents to forego their children's tonsillectomies during the Summer months.

As tonsillectomy was offered as one of the chief causes of this dread disease, and being physicians endeavoring to save human life rather than destroy or jeopardize it, we meekly accepted this theory and discontinued tonsillectomies during the Summer months. Many of us frankly doubted the validity of this conclusion as experience with thousands of tonsillectomized patients had not yielded any evidence to substantiate the theorizing of our associates.

Being a representative group of organized medicine and vitally interested in the settling of this controversial question, our present committee on the study of the poliomyelitis-tonsillectomy question was formed. Most of the members of our committee have worked tirelessly and their gratifying interest and cooperation is above reproach. To these state chairmen

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 25, 1950.

†From the Manhattan Eye, Ear and Throat Hospital, New York, N. Y.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

Cases of Poliomyelitis - By Age - Type of Disease - Operative Procedures--------Table I.

Age				Bulbar			Spinal			Mild		
	Total Cases	Operations T&A	Others	Total Cases	Operations T&A	Others	Total Cases	Operations T&A	Others	Total Cases	Following T&A	Others
0 - 5	6575	29	36	656	7	7	4261	16	25	1658	6	4
6 - 10	4897	28	43	919	13	12	2434	12	21	1544	3	10
11 - 15	2911	5	23	570	1	10	1424	2	11	907	2	2
16 & over	4864	0	60	1007	0	15	2515	0	31	1352	0	14
Total	19247	62	162	3152	21	44	10634	30	88	5461	11	30

and all the ear, nose and throat physicians who have aided, I take this opportunity to say thank you, most sincerely, for your contribution in our Society's endeavor; however, this survey has assumed gigantic proportions, and we feel it an imposition to further tax our members, who, with their heavy office and operating schedules, cannot very much longer stand this additional burden. Were we financially able to alleviate this condition, we would willingly do so, but all of our attempts to obtain financial aid were met with disapproval and rejection.

Type of Disease - Operative Procedure and Poliomyelitis By State - Year 1949.

State	Total Cases			Bulbar			Spinal			Mild		
		Operations			Operations			Operations			Operations	
	Total	T&A	Others	Total	T&A	Others	Total	T&A	Others	Total	T&A	Others
New Jersey	1086	3	3	190	1	1	588	1	1	308	1	1
North Carolina	194	0	2	19	0	0	61	0	2	114	0	0
Texas	1833	3	2	248	1	0	1131	2	1	454	0	1
New Mexico	207	0	1	35	0	1	85	0	0	87	0	0
Georgia	182	1	0	40	0	0	78	1	0	64	0	0
Oklahoma	778	3	4	165	2	2	540	1	2	73	0	0
Delaware	47	0	0	7	0	0	15	0	0	25	0	0
Indiana	181	0	1	30	0	1	151	0	0	0	0	0
Arkansas	505	5	13	48	2	1	451	3	12	6	0	0
Wisconsin	752	3	9	100	1	2	412	1	4	240	1	3
Missouri	59	3	2	6	0	1	20	3	1	33	0	0
Connecticut	646	1	4	70	0	1	556	1	3	18	0	0
Rhode Island	153	0	1	7	0	1	87	0	0	59	0	0
Arizona	107	0	0	28	0	0	54	0	0	25	0	0
District - Columbia	103	0	1	5	0	0	46	0	0	52	0	1
South Carolina	87	0	1	8	0	0	59	0	1	20	0	0
Virginia	163	0	2	18	0	0	78	0	2	65	0	0
Louisiana	125	1	3	2	0	0	85	1	3	38	0	0
Maine	377	0	4	35	0	0	197	0	4	145	0	0
Alabama	169	0	0	19	0	0	71	0	0	78	0	0
Mississippi	311	0	0	18	0	0	190	0	0	102	0	0
Oregon	225	0	3	58	0	0	138	0	3	28	0	0
Kansas	510	0	4	102	0	1	209	0	2	199	0	1
North Dakota	180	2	2	18	0	1	63	1	1	99	1	0
New Hampshire	107	0	1	19	0	1	71	0	0	17	0	0
Massachusetts	1482	4	26	276	1	6	656	1	12	530	2	6
Michigan	186	1	0	45	0	0	79	0	0	62	1	0
Washington	392	0	1	80	0	0	220	0	1	92	0	0
Idaho	358	1	4	125	0	2	178	1	2	57	0	0
New York State	2295	12	22	363	4	3	1310	5	9	822	3	10
New York City	2446	7	15	392	2	5	1109	4	6	845	1	4
Ohio	1551	4	17	273	2	6	778	1	10	439	1	1
Tennessee	199	1	2	29	0	1	140	1	1	30	0	0
Maryland	171	1	3	39	1	0	118	0	3	18	0	0
South Dakota	339	2	4	60	2	4	184	0	0	95	0	0
Florida	129	0	3	10	0	0	50	0	1	69	0	2
Nebraska	176	2	1	37	2	0	109	0	1	30	0	0
Colorado	128	0	0	26	0	0	75	0	0	27	0	0
Utah	216	1	0	55	0	0	156	1	0	5	0	0
Wyoming	113	1	1	26	0	1	58	1	0	28	0	0
	13247	62	162	3152	21	44	10634	33	88	5461	11	30

Needless to say, we have not been completely satisfied with the results of our surveys, as it is easy to see that a study nationwide in its scope has its drawbacks and does not allow for detailed and adequate research. Being cognizant of this fact, the writer, in 1947, proposed a new approach to the problem and one that might well provide a conclusive answer. Instead of obtaining through state health departments and private physicians and surgeons the records of poliomyelitis cases which had already occurred and then searching their

Table III

Distribution by Types of Involvement-Number and Percent

Year 1949.

	Total Cases	Cases Preceded By T&A Number	Cases Preceded By T&A Percent	Cases Preceded By Other Operations Number	Cases Preceded By Other Operations Percent
Bulbar	3152	21	0.7	44	1.4
Spinal	10634	30	0.3	88	0.8
Mild	5461	11	0.2	30	0.6
Total	19247	62	0.3	162	0.8

histories for evidence of previous recent tonsillectomies, it was proposed to set up a study which would answer directly the pertinent question — "Are children who have been tonsillectomized during the Summer months more likely to contract poliomyelitis than nontonsillectomized children of the general child population?"

Table IV

Distribution by Age-Number and Percent-Preceded by T&A's and Other Operations

Year 1949.

	Total Cases	Cases Preceded By T&A		Cases Preceded By Other Operations	
		Number	Percent	Number	Percent
0-5	6575	29	0.4	36	0.5
6-10	4897	28	0.6	43	0.9
11-15	2911	5	0.17	23	0.8
16&over	4864	0	0.0	60	1.2
Total	19247	62	0.3	162	0.8

As outlined, the study would follow several thousand children who would ordinarily be tonsillectomized during the months of June, July, August and September. So that varying epidemic characteristics of the disease would be properly balanced, it was planned to select approximately 20 hospitals throughout the United States to cooperate in the study. At the time of admission to the hospital, there would be attached to the regular chart a special card listing pertinent information regarding age, sex, color, date of tonsillectomy and space for comments for a weekly one-month follow-up study. Following tonsillectomy and the patient's discharge from the hospital, the card would be turned over to special social service workers for follow-up at weekly intervals to determine whether or not poliomyelitis ensued. Such a study, properly supervised and tabulated, would give conclusive evidence

Table V
Bulbar Cases Following Tonsillectomy

Locality	Age	Number of Days Following T&A
New Jersey	6	Day of onset
Texas	2	6 days
Oklahoma	5	14 days
Oklahoma	5	13 days
Arkansas	3	Day of onset
Arkansas	9	14 days
Wisconsin	10	15 days
Massachusetts	12	14 days
Ohio	3	8 days
South Dakota	6	22 days
South Dakota	7	14 days
Nebraska	6	21 days
Nebraska	8	9 days
Maryland	6	14 days
New York State	7	10 days
"	6	7 days
"	6	12 days
"	9	6 days
New York City	5	6 days
"	5	9 days
Ohio	8	14 days

whether the incidence of poliomyelitis in recently tonsillectomized children was significantly greater than the normal incidence of the disease in the general child population.

Table VI

Deaths from Poliomyelitis

Age Group	Cases of Poliomyelitis	Number in which Death Resulted	Case Fatality Rate
0-5	6575	195	2.9
6-10	4897	228	4.6
11-15	2911	152	5.2
16&over	4864	474	10.0
Total	19247	1049	5.4

Having taken all aspects of this problem into consideration, we have decided that we already have at hand enough material from our surveys to formulate an opinion and that the continuance of this survey would only be superfluous data. Narrowing our fields somewhat, we feel that were we to investigate the cases in the City of New York, *i.e.*, comparing the tonsillectomized and nontonsillectomized child population, more concise data could be obtained and follow-up more detailed and advantageous. This I plan to do and if anyone from the larger centers would care to volunteer his services and follow my proposed procedure, I would be delighted to discuss the matter with him at further length.

Table VII

Bulbar Type of Poliomyelitis

Year 1949.

Total Number Cases Poliomyelitis------19247
Total Number Cases-Bulbar Type--------3152 or 16%
Bulbar Cases Following T&A------------21 or 0.7%
Bulbar Cases Following Other Operations----44 or 1.4%
Bulbar Cases Following No Operations------3087 or 97.9%

Several of our state chairmen's reports, unfortunately, could not be incorporated in our standard charts as they, the chairmen, found it impossible to obtain the necessary data that would conform to our pattern of tabulation. This was due to either the lack of cooperation on the part of the state departments of health or to the respective states' high rate of incidence of poliomyelitis which in these few states was so unusu-

Diseases and Accidents Preceding Onset of Polio

Table VIII

Year 1949.

Tooth extraction
pregnancy
appendectomy
delivery
episiotomy
myringotomy
chicken pox
cut foot
whooping cough
incision-axillary abscess
head injury
perineal abscess repair
miscarriage
herniotomy
cellulitis
kidney stone-removed
lacerations of hip-sutured
epistaxis-cauterization
fractured bones
removal-ingrown toenail
herniorraphy
recession
gum boil
vaccination
nail in foot-tetanus shot
shot in finger-tetanus "
incomplete abortion
pneumonia
circumcision
auto accidents
ear and sinus infection
teeth filled
lacerations
tracheotomy for croup
mangled hand-repair
falls
cholecystectomy
dilitation and ourettage
Incised boil-buttocks

ally high that it would have necessitated additional secretarial workers. Due to circumstances beyond our control, this situation could not be avoided.

Table IX

BULBAR CASES FOLLOWING TONSILLECTOMY

STATE	TOTAL CASES	BULBAR	STATE	TOTAL CASES	BULBAR
ARIZONA	422	0	NEBRASKA	404	0
ARKANSAS	232	0	NEW JERSEY	1148	0
CALIFORNIA	9177	0	NEW MEXICO	100	0
COLORADO	316	0	NO. CAROLINA	1943	0
CONNECTICUT	1081	0	NO. DAKOTA	67	0
DELAWARE	2936	0	OHIO	2545	0
D OF C	260	0	OKLAHOMA	373	0
FLORIDA	698	0	OREGON	484	0
GEORGIA	599	0	PENNSYLVANIA	7530	0
ILLINOIS	2671	0	RHODE ISLAND	964	0
INDIANA	1568	0	SO. CAROLINA	598	0
IOWA	975	0	TENNESSEE	946	0
KANSAS	140	0	TEXAS	1434	0
LOUISIANA	837	0	VIRGINIA	1537	0
MASSACHUSETTS	4154	1	WASHINGTON	115	0
MICHIGAN	2087	0	WISCONSIN	354	0
MINNESOTA	313	0	N.Y. STATE	10943	1
MISSISSIPPI	300	0	CANADA	211	0
MISSOURI	806	0	HAWAII	72	0

TOTAL(1949)........................... ....61340.................2

Previously Reported(1948)..............................35039

Totals 1946-1947-1948-1949.............................96379

Total number cases following tonsillectomy..........................7 or .000073%

CONCLUSIONS.

Having completed this four-year study of 36,678 cases of poliomyelitis as well as 96,379 cases of tonsillectomies, we fail to see any causal relationship existing between poliomyelitis and tonsillectomy nor, up to and including the present time, have we read any published reports from other sources that offered any conclusive proof that tonsillectomy does predispose to bulbar poliomyelitis.

As we have said in previous years, we see no reason why tonsillectomy should be indefinitely postponed simply because the summer months are the months wherein poliomyelitis is prevalent; however, we do not advise any elective surgery being done during any epidemic, regardless of its nature.

STATE CHAIRMEN
POLIOMYELITIS SURVEY
YEAR 1949

Alabama	Gilbert E. Fisher
Arkansas	Paul L. Mahoney
Arizona	Joseph M. Kinkade
California	Howard P. House
Colorado	James P. Rigg
Connecticut	Edward R. Roberts
Delaware	William M. Pierson
Washington, D. C.	Samuel L. Fox
Florida	Charles C. Grace
Georgia	Russell B. Burke
Idaho	Roy La Vern Peterson
Illinois	Oliver E. Van Alyea
Indiana	Carl H. McCaskey
Iowa	Clair M. Kos
Kansas	William P. McKnight
Kentucky	William C. Wolfe
Louisiana	James W. McLaurin
Maine	Fred T. Hill
Maryland	Samuel L. Fox
Massachusetts	Robert L. Goodale
Michigan	J. Louis Dill
Minnesota	Lawrence Boies
Missouri	John C. Howard
Mississippi	Edley H. Jones
Montana	Voss Harrell
Nebraska	Lyman H. Heine
Nevada	Voss Harrell
New Hampshire	John A. Murtagh
New Jersey	Edgar P. Cardwell
New Mexico	Franklin P. Schuster

New York City	W. Wallace Morrison
New York State	Benjamin M. Volk
North Carolina	Watt W. Eagle
North Dakota	Nelson A. Youngs
Ohio	Harry C. Rosenberger
Oklahoma	William L. Bonham
Oregon	Lewis W. Jordan
Pennsylvania	Harry P. Schenck
Rhode Island	Nathan Bolotow
South Carolina	Richard W. Hanckel
South Dakota	Paul G. Bunker
Tennessee	Guy M. Maness
Texas	Franklin P. Schuster
Utah	James P. Rigg
Vermont	De Forest C. Jarvis
Virginia	Elbyrne G. Gill
Washington	Raymond A. Lower
West Virginia	Unable to obtain chairman
Wisconsin	Thomas L. Tolan
Wyoming	James P. Rigg

2. That these changes may be either of two types: *a.* red, dry, irritated and desquamative or "chapped"; *b.* pale, wet and boggy,

3. That patients deficient in thyroid hormone may have an increased tendency to nasal infection and nasal allergy which can be corrected (or at least influenced) by the administration of thyroid extract alone,

4. That headache and nasal obstruction are prominent symptoms,

5. That the basal metabolic rate is helpful but not infallible in distinguishing cases suitable for treatment with thyroid extract, and

6. That the basal metabolic rate is not a reliable prognostic index but that in all cases the response of the patient is more useful, and determines the optimum dosage as well.

Some of the cases here under discussion belong in the group of extreme deficiencies, but on the whole they are found in individuals who would receive little or no thyroid treatment from the physician on the grounds of findings outside the nose.

Briefly stated the present findings are as follows:

Total cases	130
First suspected of hypothyroidism on basis of nasal appearance alone	66
Previously diagnosed	52
Incomplete	4
Suspected of hypothyroidism but reported plus B. M. R. (Seven of these were males)	8

AGE.

	Females	Males
1-10 years	1	0
11-20 years	4	3
21-30 years	21	5
31-40 years	29	11
41-50 years	27	7
51-60 years	7	2
61-70 years	4	1
	93	29

From what is known of the effects of thyroid deficiency on other tissues, such nasal symptoms might be expected.

Means[10] devotes a chapter to "Thyroid Administration in Diseases of Other Than Thyroid Origin — which is to say the use of the thyroid hormone for its nonspecific drug action" in which he cites references to changes in the adrenals, the kidneys, the blood, the stomach, the nerves, the intestines, the genital organs, the skin, the heart, the ear and the eye! This alone might be regarded as presumptive evidence of changes in the nose, an organ characterized by the most sensitive of neurovascular, glandular and epithelial tissues.

"Broadly speaking," writes Means, "the thyroid hormone has to do with the rate of growth and differentiation of tissues, with the rate of energy exchange, with the metabolism of proteins, carbohydrates, fats and lipoids, salts and water, with the irritability of various tissues and with the activity of other endocrines." Is it conceivable that the nose could escape all this?

Byrom[1] hypothesizes a laxity in the intercellular cement substance as the result of thyroid deficiency. Such a condition, if it exists, must have important consequences in the nasal epithelium, the integrity and impermeability of which is regarded as the first line of defense against pathogenic organisms. If such a weakened intercellular substance were to permit even occasional bacteria to enter which would have to be dealt with by the underlying secondary defenses it might well result in the low-grade inflammatory reaction which I have described as a "chapped" membrane.

Means states further that "while the thyroid hormone increases the demand for oxygen by the tissues, it likewise provides the wherewithal for this demand to be satisfied" (extra blood supply by more rapid heart action, increased stroke volume of the heart, peripheral vascular dilatation). Conversely it is known that when oxygen consumption, and therefore heat production, is reduced body temperature is maintained by shutting down the heat loss: peripheral circulation is reduced, sweating stops, muscular activity is slowed

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Chavanne,[2] in 1936, injected thyroxin into the circulation of a dog and demonstrated a drop in the weight of the nasal secretions for a short period. This could be repeated at will. Unfortunately a second dog failed to react in like manner. He then removed the thyroid glands from these two dogs, increasing the nasal drainage which could again be held in check by the injection of thyroxin.

Animal experiments have not been done by us simply because no method suggests itself by which conditions encountered in the clinic can be reproduced. Total thyroidectomy obviously will not serve. Partial thyroidectomy fails because the remaining tissue undergoes hyperplasia, preventing a fall in the metabolic rate.

Most informative have been the selected cases referred to,—those known to be deficient in thyroid hormone, free from purulent sinusitis, atrophic rhinitis, occupational irritants, polyps and demonstrable allergies. These range all the way from a dry, uncomfortable nose in an old woman to a bloody, ulcerated membrane incapacitating a college student in whom all manner of local and supportive treatment had failed.

At present writing evidence of characteristic changes in the nasal mucosa arising from thyroid insufficiency rests upon three observations:

1. That in 87 per cent of cases suspected of such deficiency on the basis of nasal findings alone, a subnormal metabolic rate has actually been present,

2. That a high percentage of cases show unquestionable response to specific therapy, and

3. That in selected uncomplicated cases pronounced improvement followed treatment with thyroid substance alone after other means had failed, relapse occurring when the medication was withdrawn.

Quite probably the influence of the thyroid in many of these cases is modified by the activity of the related glands, more particularly the adrenal and the pituitary. Although recent comprehensive reports on Adrenocorticotrophin (ACTH)[13] and

Cortisone[9] make no mention of the nasal mucosa, there can be little doubt that this highly neurovascular tissue reacts similarly to others described and that the hormones of the thyroid and the adrenal cortex, here as elsewhere, are antagonistic to one another. This may account for those cases in our series which, though having apparently the same characteristics as the rest, failed to respond to thyroid extract.

Recognition of this condition depends at present chiefly upon the awareness of the rhinologist of its existence. Variable and vague though the symptomatology may be, the diagnosis is simple. A low basal rate is presumptive evidence; a therapeutic test conclusive.

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Beaumont Building.

THE TREATMENT OF SURGICAL SHOCK.*

WILLIAM G. MEALS, M.D. (by invitation),
Los Angeles, Calif.

In a review of 32,000 anesthetics given by our group in the past eight years we have on record only one case within your specialty requiring such therapy. This may be because so many of your major procedures are performed under local methods; hence, we do not see all of your work. Otolaryngological surgery is slowly changing. Radical head and neck surgery is being performed by your specialty so that more and more men are being confronted by the problem of surgical shock. Similarly, extensive plastic surgery is being perfected. While these are not particularly shocking procedures, they frequently involve a long anesthetic which may predispose to shock and necessitate preventive and therapeutic shock measures. Be that as it may, surgical shock can and will appear when least expected and for this reason everyone practicing medicine must be ever aware of its treatment.

Shock is a disturbance of fluid balance resulting in a peripheral circulatory deficiency which is manifest by a decreased volume of blood, reduced volume flow, hemoconcentration and renal functional deficiency. Capillary tonus and contractility are controlled by levels of oxygen need and metabolic activity. Anoxia, toxins and the products of metabolism produce relaxation and increased capillary permeability, thereby giving rise to more endothelial surface by opening more capillary beds. Fresh arterial blood contains a hormonal substance, probably arising from the pituitary gland, which causes capillary contraction. Control of the circulatory cycle of the capillary bed is thus maintained in direct proportion to the functional

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 25, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

activity of the cells supplied. This is the most important physiologic concept to remember in shock. The patient literally bleeds into his own capillary beds, causing a disparity between the blood volume and the vascular bed.

Shock has been classified on an etiologic basis into three types: hematogenic, neurogenic and vasogenic. Hematogenic shock is characterized by loss of circulating blood volume, neurogenic shock by reflex loss of vasomotor control, and vasogenic shock by dilatation of capillary vessels through the direct action of toxins on their walls. Two mechanisms, then, may produce shock: increased intravascular space, or loss of circulating blood volume with hemoconcentration. The former is primary, the latter secondary shock.

Primary shock is best exemplified in syncope, or fainting, where sudden loss of vasomotor tone with reflex slowing of the heart results from sudden painful physical or psychogenic stimuli. It is generally of short duration and recovery results from spontaneous return of the sympathetic vasomotor control. The arterioles and capillaries dilate suddenly, producing a tremendous increase in intravascular space. This type of shock is seen when local anesthetic agents are too rapidly absorbed or unintentionally injected into the blood stream.

These occurrences are quite common, and a brief word concerning their care and prevention might not be amiss. Fainting from any cause needs little therapy. Lowering the head, smelling salts and release of binding clothing are sufficient. The main therapy is prevention. This is done by reassurance and telling the patient when and if a certain procedure is to be painful. For our convenience instruments are often placed so as to present a terrifying picture to the patient. Any simple method of rendering them less conspicuous alleviates this problem. The patient who has absorbed too much local or received an accidental intravenous injection requires more care. An intravenous barbiturate injected early will quickly calm the garrulous and excitable patient and also provide protection against more serious reactions such as convulsions. In these more serious cases oxygen inhalations are essential as death results from anoxia. An intravenous barbiturate, suction and a mechanical airway should be available immediately whenever local anesthetics are being used in the care of a patient.

In secondary shock depletion of blood volume, regardless of the cause, is the initiating mechanism. Circulatory failure results from diminished cardiac output due directly to an inadequate venous return. Peripheral arterial resistance may be adequate to maintain blood pressure for some time in spite of an obviously failing venous return. Eventually this peripheral resistance will weaken and the blood pressure may then fall precipitously. All this time the capillary bed is opening up more and more channels, literally swallowing the erythrocytes; capillary stasis follows; anoxia occurs, causing further capillary beds to open. The capillary dilatation, the anoxia, the slow flow of blood, the lowered blood pressure all tend to initiate a vicious cycle, which if not stopped will surely and unrelentingly lead to death. Lowered metabolism and acidosis develop slowly and parallel the systolic blood pressure. The kidney function is deranged in shock. There is a reduction of blood flow and hemoconcentration, which the body resists by retaining fluid rather than letting it filter through the kidneys. Some patients, apparently well recovered from shock, die in uremia from failure of the kidneys to function properly after the shock.

Surgical shock, whether occurring during or after surgery, results from the combined effects of the disease, necessitating surgery, the anesthetic, the absorption of substances from damaged tissue, hemorrhage, and fluid loss from the surgical area. The emotional response of the patient is an important initiating stimulus. Last, but not least, the judgment, and skill of the surgeon and the anesthetist are important contributory stimuli. Surgical shock, then, includes both primary and secondary shock and is a combination of the three etiologic types: hematogenic, neurogenic and vasogenic.

Dr. Crile made a most astute observation when he said, "The best treatment for shock is prevention." This holds true to the letter and, fortunately, such practice is most common today. Preventive care is preoperative care. Serious attention is given to nutrition and general health even weeks before surgery. Minor ailments and dietary deficiencies are corrected. Anemia is treated, even to the extent of several transfusions, days before surgery. Patients are frequently hospitalized well beforehand to attend to proper nutrition, hydration and medication prior to operation. In emergencies, shock is treated first so that the individual can better withstand the insult of surgery. The better the physical condition of the patient, the smoother will be the operative and postoperative period.

Before coming to surgery proper premedication is essential to allay apprehension, worry, fear and anxiety. A well-premedicated patient is a far safer operative risk. Proper acquaintance and judgment in handling such medication is essential as overdosage may play a part in the development of shock through depression and anoxia.

During surgery, trauma and fluid loss should be kept to a minimum; anesthesia should be neither too light nor too profound. When surgery of any magnitude or duration is contemplated, intravenous fluid should be given from the onset; not so much because of its need, but rather so that in case of emergency a ready portal of entry for immediate replacement therapy is instantly available. Blood, plasma or some suitable replacement should be attainable within a short period of time. These precautions, so universal today, have resulted in a marked decrease in the occurrence of shock during surgery or its immediate postoperative course.

Now, if surgical shock does appear, how is the condition to be treated? Here the judgment of the surgeon and the anesthetist plays a rôle of prime importance. The cause for the onset of the condition must be ascertained at once. Is it due to the anesthetic, reflex phenomena from surgical manipulation, fluid loss, gross or concealed hemorrhages, or to the disease for which surgery is being done? Occasionally one cause can be identified as the predominate stimulus, but by and large the inciting factor is a well amalgamated combination of stimuli. The important part of treatment is early recognition and instant active therapy to reverse the physiological changes taking place, for as shock progresses its treatment becomes increasingly difficult. Because of the inefficient circulatory system, it is imperative that replacement fluids be introduced directly into the blood stream, either by intravenous or bone marrow injections. Generally speaking, the more closely the fluid given resembles that which has been lost, the more satisfactory the effect.

Intravenous fluids in the form of electrolytes are only mildly satisfactory. If the shock is early, if it is due to anesthesia, or if no better replacement is available, they are useful as temporary measures and generally are used to supplement the daily fluid supply. As a rule, surgical patients are deprived of fluids for eight hours preoperatively; hence, their need for fluid. Their value is mainly to maintain body hydration and as a preventive rather than a therapeutic agent. Such fluids leave the blood easily and cause only a temporary and transient increase in blood volume. Animals, experimentally bled or placed in severe shock, show no increase in survival rate when treated with the electrolytes as replacement fluids than those receiving no replacement whatsoever. Hence, such fluids are used mainly as preventive measures, or in early treatment while awaiting the arrival of more satisfactory replenishments for the blood volume.

In the majority of cases of clinical shock, plasma or blood is necessary for effective restoration of the circulatory deficiency. If little whole blood has been lost, plasma is the obvious choice for replacement. The amount to be given is not dependent upon any calculable formula or routine, but must be given until the patient has returned to a satisfactory condition. Plasma comes in frozen, liquid or dried forms, all of which are equally effective and completely interchangeable. Little time is utilized in preparing the solution and no tests are necessary before it can be administered. Its use has fallen into some disrepute because of the occurrence of a significant number of cases of homologous serum hepatitis. Plasma is prepared from pools, and one donor can infect the entire lot; however, where active therapy is needed, treatment should be given in spite of the chance of this complication. Experimentally, animals placed in lethal shock, either from hemorrhage or trauma, show nearly as high rate of recovery as those treated with whole blood. This is especially true in animals when the shock was caused by trauma. The rationale behind the use of plasma is obvious:

1. It restores the blood volume by the same type of fluid that was lost.
2. It does not escape into the tissue readily unless considerable endothelial damage has occurred.
3. It exerts a normal type of osmotic pressure tending to draw fluid back from the tissues into the blood stream, thereby counteracting the mechanism by which shock progresses.
4. It restores directly and immediately the plasma protein deficiency which follows incident to shock.

In hemorrhagic shock whole blood is the most logical and efficient replacement fluid. The amount to be utilized depends upon the amount lost and upon the patient's response to replacement. Most failures in treatment are due to inadequate refilling of the vascular bed. Too many doctors tend to sit back and relax after a patient has had one transfusion. Replacement must continue until the blood pressure, pulse, color and hematocrit determination are back to normal levels. Treatment must not be discontinued as soon as a normal state is approached, as often there is a compensatory rise, even above normal levels, which may be followed by a return to the shock state unless therapy is watchfully continued. In other words, the activity of treatment can be diminished, but should not be terminated until the normal state has been maintained for a considerable period. In some instances of hemorrhagic shock, surgical intervention may be the only method of terminating the hemorrhage. Enormous quantities of blood may be necessitated and such *quantities can, and should, be given.* The patient should receive blood until he is out of the shock state before he is taken to surgery and should continue to have it until surgery is completed. This is perhaps one of the greatest lessons learned in the last war. Plasma can be used along with the blood, or as a temporary substitute, so long as no more than 1 liter is given. In hemorrhagic conditions an overdosage of plasma may result in permanent hemodilution, secondary anemia and diminished oxygen carrying power of the blood.

In giving fluids, certain routine measures should always be followed. A vein should be chosen which is accessible, yet which will permit the patient some freedom of motion and allow free movement of the patient with little danger of its dislodging and infiltrating the surrounding tissue. The needle and tubing should be fitted firmly in place. The needle should be of sufficient size so that any replacement fluid can flow easily and quickly through it. The usual size is either an 18 or 19 gauge. Such precautions allow for permanence and for rapid or slow flow of the fluid. In extreme cases a larger needle with a trocar can be introduced into the vein, either directly or by surgical cutdown. Such a needle can be left in place as long as necessary. The speed of replacement is important. If shock is severe, one pint of blood or fluid can be injected in from three to five minutes, while following fluids can be allowed to enter at a variable rate, depending upon the patient's response.

The giving of transfusions of blood is more complicated than giving other fluids as the matter of blood types occurs. If the need for transfusion is anticipated, this, of course, presents no problem as the proper type blood will be ready in surgery. If the need of some is foreseen in surgery, it can usually be ordered well in advance, so that the laboratory can have it ready. If emergency need should arise, however, type O blood, that is, RH — can be given to females and type O — RH+ to males. In many large clinics and institutions this procedure is frequently almost routine and the incidence of reactions is small. Transfusion reaction is on the decline, but if symptoms or signs should occur, the transfusion should be stopped immediately.

The importance of the use of oxygen inhalations cannot be overemphasized in the treatment of circulatory deficiency. In oxygen deficiency there is a decrease in alkali reserve with a resulting slide toward acidosis. Oxygen lack results in incomplete combustion and the formation of abnormal catabolic products. This gives a disturbed electrolytic balance between the cells and the extra cellular fluid. Most important, lack of oxygen relaxes and renders more permeable the capillary endothelium. This produces a self-perpetuating chain of events, causing the circulatory deficiency to progress in the manner of a vicious cycle. With these considerations in mind, it is little wonder that the value of oxygen in the treatment of shock is being stressed more and more. Experimentally, dogs in shock maintain their blood pressure at a higher level and live longer if given oxygen inhalation. Similarly, animals given oxygen inhalations tolerate more severe hemorrhages than do the controls. Oxygen can be given via tent, mask or catheter. Of all methods, the catheter is perhaps most comfortable and efficacious. If under anesthesia, the patient is given pure oxygen with no anesthetic agent. Its simplicity of administration and its availability make it the easiest form of medication in shock, but unfortunately the most neglected. No case treated for shock is being treated adequately unless oxygen is administered immediately. It is frequently the first treatment received because of its instant availability.

Any other treatment of shock is of little value. Drugs, by and large, are of little avail. Adrenalin-like drugs, especially ephedrine or neosynephrine, are of use in cases of shock due to spinal anesthesia and possibly in very early shock. Adrenalin is contraindicated. The circulatory system is already in a state of extreme vasoconstriction down to the finest arterioles, hence the use of these drugs is needless; in fact, shock can be induced by repeated use of such drugs. The function of the adrenal cortical hormone is to maintain the normal impermeability of the endothelium and the cells. The use of eschatin, a cortical extract, or synthetic desoxycorticosterone may have some use. Such use would be mainly preventive as experimental evidence shows that its use may make animals more impervious to shock. Stimulants and analeptics have been used, but the rationale behind their use is not evident. They in no way affect the tonus or permeability of the capillaries nor do they increase the efficacy of a failing circulation.

Mechanical methods to facilitate the return flow of venous blood from the periphery have been tried. The reasoning behind such action is not sound, as the stagnation of blood is not on the venous side but rather in the capillaries. External pressure or gravity have no effect upon the capillary bed.

There are several available substitutes for blood or plasma that are quite efficacious and produce prolonged effects. Such replacements can be advantageously utilized if the desired replacements are unavailable immediately. In mild cases, their use alone may be satisfactory treatment. To be satisfactory as a blood or plasma substitute, a fluid must meet certain definite specifications. Its molecular size must be such that it will not escape from the vessels. Its viscosity and osmotic pressure must approach those of blood. It should be isotonic with the contents of the erythrocytes. It must be nonantigenic and innocuous. Of the innumerable substitutes that have been presented, those proving most efficacious have been acacia, pectin, peristan and dextran. Gum acacia, the oldest, given in physiologic saline is well tolerated and has definite beneficial effects; however, it has caused serious reactions and is not readily eliminated, forming deposits in the liver and spleen. Pectin, an acid derivative of citrus fruits, was introduced in 1941. It is nontoxic and nonantigenic, and well handled by the system, being eliminated within 72 hours. While it causes some pseudoagglutination of the erythrocytes, its value more than compensates for this. Partisan is a synthetic polyvenyl pyrollidin developed by the Germans and used extensively by them in World War II when they could not supply sufficient amounts of blood or plasma for their wounded. It is inactive in the body, remains actively in the circulation for two or three days, and is completely eliminated. Only one case of drug reaction has been reported after thousands of instances of its use. The newest, and probably what will be recognized as the best substitute is dextran, a product of Swedish research. It is a polysaccharid of high molecular weight. It is used in a 6 per cent solution in normal saline. It is eliminated slowly over a five-day period, giving a prolonged period of action. No drug reactions of any severity have been noted following its use. These substitutes are only substitutes, and in cases not showing immediate results all efforts should be made to obtain blood or plasma.

Shock is a state of circulatory deficiency, which, if not treated at its very onset and treated diligently, will progress steadily and relentlessly to a fatal ending. Its treatment consists in the main in adequately replenishing the circulating blood volume with blood, plasma or a substitute while concomitantly furnishing oxygen to oxygenate the blood adequately. If these two factors of treatment are carried out with watchful care, the shock will generally be reversed and a satisfactory outcome will follow.

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ALLERGY — EFFECTIVE TREATMENTS USED IN OTOLARYNGOLOGY.*

MARVIN FISHER JONES, M.D.,
New York, N. Y.

Otolaryngologists need a simple, practical program by which they may successfully treat their allergic patients. In the main, the procedures recommended in this paper have been used by me for my nonseasonal allergic patients over the past 30 years. Their greatest value lies in their simplicity and their effectiveness. Most patients who have recurrent attacks of sinusitis can be permanently cured. For some patients who have chronic sinusitis, sinus surgery will still be necessary. Even for the diminishing number of patients who require sinus surgery there is an added assurance of permanent good health if antiallergic procedures are used following their operation.

There is a popular expression, "Once a sinus patient, always a sinus patient." Perhaps this has been true in the past, but at present it is definitely false. When there is intelligent cooperation by patients in helping to cure their allergies and indicated surgery its expertly performed, most protracted cases of sinusitis can be permanently cured.

We must accept as a fact that the incidence of sinus surgery has decreased. This decrease had started before the use of sulfa drugs and antibiotics. In fact, these newer drugs have been somewhat disappointing in their effects on chronic sinusitis. The decrease in the number of operations on the sinuses was due to several causes. Dissatisfaction with the results obtained by surgery was felt by both patient and doctor. Ill advised or inadequately performed surgery caused patient

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 27, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

dissatisfaction. Failure to recognize allergy as a cause of recurrent sinusitis led to repeated operations.

Allergy is now becoming generally recognized as a most important cause of sinusitis, and the successful treatment of allergies has had its effect in the lessened need for sinus surgery. Until recent years, however, the importance of that relationship was not generally recognized. It should again be emphasized that proper care of the allergic patient in the future will still further reduce the need for sinus surgery. The effectiveness of our effort to prevent sinusitis will depend upon the alertness of the otolaryngologist in recognizing diagnostic symptoms of allergy and his efficiency in treating the cause. We must not only be alert to the allergic causes of symptoms but we must also recognize the proper time for surgical intervention. In order to prevent recurrences of sinusitis following operations on the sinuses, we must also cure the allergies which caused the recurrence of symptoms.

I have tried many methods in order to create a satisfactory diagnostic and treatment policy over these many years. Experience has convinced me that the otolaryngologist who has been adequately trained in the diagnosis and treatment of allergy will obtain the greatest percentage of permanent cures for his nonseasonal allergic patients. The next most effective arrangement is through an association with an allergist on an equality basis. Such an arrangement is apt to prove unsatisfactory if either the otolaryngologist or the allergist is in a position subordinate to the other.

My total program for the care of an allergic patient can be outlined as follows:

1. Dietary Selection.
2. General Health Measures.
3. Correction of Emotional Instability.
4. Immunization and Antihistaminic Drugs.
5. Elimination of Contacts.
6. Surgery.

The combined use of these six methods has produced maximum results. Nos. 1, 2, 4 and 5 will be discussed in this paper.

Food is the most important single cause of allergy in those patients who have consulted me as an otolaryngologist. Of course, I am aware that this statement is contrary to the respected opinions of other authorities. Because I believe most patients who consult the otolaryngologist regarding their symptoms are food-sensitive patients, the subject of proper food selection will be the major theme in my presentation. Again I must state that I am limiting my remarks to nonseasonal allergy.

The allergic ingestants are usually of a retiring nature and in this respect they differ radically from the seasonal allergies characterized by dramatic explosions. Likewise, in using skin tests for diagnosis, the foods respond in an unpredictable manner, while skin tests for the seasonal varieties are more reliable. Food allergies are generally more difficult to detect. The most useful method of determining which foods are responsible for symptoms is a clinical one. The purpose of selecting foods ordinarily believed innocuous when ingested is to relieve the symptoms caused by other foods as soon as possible. Always recommend allergen-poor foods in adequate amounts and variety in order to completely fill the nutritional, vitamin, mineral and appetizing requirements. All patients must have a balanced, attractive diet in the beginning. Later, any foods the patient will tolerate are added. Any supplements to the original diet are made after a six weeks symptom-free period. If one must attach a name to this policy, "A Dietary Selection Method for Diagnosis and Treatment" might be appropriate. In addition to my own experience in choosing proper foods, I have made free use of information published by such authorities as Rowe, Vaughn and Hansel, and the personally communicated contributions of Blake F. Donaldson. One of the greatest hindrances to a proper diet selection is the unreliability of lists based on skin reactions. New lists must be made designating foods which cause clinical reactions. Such a list should also include the symptoms caused by each food. A new list is under process of compilation by me which will be based on clinical reactions. There is great need of many such long-range reports. The greatest difficulty seems to be a differentiation between allergic reactions and indigestion. There is some evidence to indicate that an allergic reaction is one and the same with indigestion. Strangely enough, the tables of foods causing complaints which have been mentioned in McLester's "Nutrition and Diet in Health and Disease" parallel closely the usual lists of foods which cause allergic reactions (Tables 5, page 145; 6, page 146; 7 and 8, page 147; and Table 9, page 148). Practically all the foods named by McLester as causing indigestion may also be classed as allergic.

The title "Selective Diet" indicates a positive approach and does not carry the inference of deprivation. This simple method recommends one complete diet selected primarily because of its food safety and, second, its clinical adequacy. According to an increasing number of authorities, such a diet should be of a high-protein type. The selection of a proper protein now becomes important.

Quoting Norman S. Moore, of Cornell University, in his article, "Facts About Nutrition," *New York State Journal of Medicine,* May 1, 1950, "Overall resistance to infectious diseases, which is, after all, the subject of most practical concern to the physician, appears to depend upon adequate nutrition, and especially protein nutrition." To quote again from McLester's "Nutrition and Diet in Health and Disease, page 188, year 1949, "Meat takes a prominent place in the dietary because of its flavor, the stimulating influence of its extracts and the high biologic value of its proteins. As a good source of vitamins also, notably thiamine and riboflavin, it is receiving increasing recognition . . . the conclusion there reached that 100 gm. of protein, more or less, represents the optimum daily intake for the average adult."

Three main sources of protein are available, *i.e.,* eggs, milk and meat. The relative values of these proteins are stated by McLester ("Nutrition in Health and Disease," page 188, year 1949) : "The proteins of meat are surpassed in nutritive quality only by those of milk: they are equalled by the protein of eggs." The proteins of milk and eggs are not available for our use because these foods stand first in the list of allergic food offenders. Fortunately, we are still able to use the highly satisfactory meat protein as a basis for our "Selected Diet" for allergic patients. We have selected meat protein as the foundation on which to build our diet, and we have ample authority to warrant the statement that we are on sound therapeutic ground. Pork and beef are occasional causes of allergic reactions, and these meats must sometimes be replaced by fowl or fish proteins. No meat causes symptoms as frequently as eggs or milk. Other nonallergic foods are grouped around meat in order to furnish a diet with adequate calories, vitamins and minerals. Believing that appetite and a zest for food is an important factor in proper nutrition, a variety of attractive foods is selected. Meat with its normal fat is the main part of each of the three daily meals.

At the end of a six weeks symptom-free period, foods are constantly added. If any food produces a recurrence of symptoms, that food is again removed from the diet. Six weeks to two months later the same food is again added, and if there is a recurrence of symptoms, that food is permanently removed from the diet.

Another method is occasionally tried in which forced feeding of some individual food is ordered in order to precipitate an attack. In case an attack is precipitated, the food is removed and a similar procedure is again tried at a later date. The occurrence of a second attack following ingestion prohibits further use of the offending food. The following list of foods is grouped according to the degree in which they are responsible for allergic reaction. This list is under constant revision and the changes are based on clinical experience.

All authorities agree on certain foods as being the ones most apt to cause allergic symptoms. These foods are:

1. Milk and milk products.

2. Wheat and wheat products.

3. Eggs.

FOOD LIST.

H	A	L
Wheat	Barley (Malt)	Rye (Ry-Krisp, Horlamus Rye Bread)
Onion	Corn (Karo Syrup, Mazola Oil)	Oats
Garlic	Bananas	Rice, Wild Rice
Leek	Asparagus	Popcorn
Turnip and Rutabaga	Cornstarch	Sorghum, Cane Sugar
Cabbage and Kale	Rhubarb	Tapioca
Mustard	Buckwheat	Cocoanut
Radish	Spinach	Date
Nuts	Cauliflower, Broccoli	Oleomargarine (Gold Dot)
Strawberry	Brussels Sprouts	Pineapple
Apple	Kohlrabi	Mulberry
Orange	Horse Radish	Fig
Cottonseed	Quince	Chard—Swiss
Chocolate-Cocoa	Pear	Chard—Kale
Spices and Seasonings	Peach	Beet
Tomato	Nectarine	Currant, Gooseberry
Peppers	Apricot	Raspberry, Blackberry
Cucumber	Lentil	Dewberry, Loganberry
Watermelon	Bean	Plum, Cherry
Cantaloup	Tangerine	Water Cress
Pepper (black and white)	Grape	Lentil, Bean
Honey	Raisins	Soy Bean
Eggs	Grape Vinegar	Olive
Ice Cream	Okra Gumbo	Lemon
Clam	Celery	Grapefruit
Lobster	Irish Potato	Lime
Shrimp	Eggplant	Sugar—Maple
Prawn	Chichle	Tea
Crab	Mussell	Carrot (vit. A high)
Pork, fresh or smoked	Scallops	Parsnip
Beef, Veal		Parsley
Jello contains pork and beef		Avocado (Alligator Pears)
Milk (small amount of allergin in butter)		Huckleberry
Cheese. Milk allergins may tolerate cheese		Cranberry
		Sweet Potato—Yams
		Squash, Pumpkin, Gourd
		Lettuce, Endive, Chicory
		Salsify (Oyster Plant)
		Artichoke
		Dandelion
		Guava, Vanilla
		Oyster
		Soy Products

Legend:
H = a high percentage of allergic reaction.
A = an average percentage of allergic reaction.
L = a low percentage of allergic reaction.

4. Shell fish.
5. Chocolate.
6. Nuts.

Such foods must be completely eliminated during the trial diagnostic period. They may be tried singly in large amounts as a diagnostic procedure.

The list of foods now recommended for the trial diagnostic period is the same list which is used for treatment.

HIGH PROTEIN MEAT DIET.

Meats: Meat is to be eaten three times a day. Pork, beef, veal, lamb, mutton and liver (three meals a week with kidneys or sweetbreads as occasional alternates). No smoked or processed meats. No fowl or fish.

Vegetables: Sweet potatoes, raw carrots, beets, artichokes, and alligator pears, brown rice and wild rice, soy beans.

Fruits in Season: Grapefruit and lemon, lime, dates, figs, dried prunes, dried apricots, pears, plums.

Tapioca.

Fluids: Tea with lemon and sugar, lemonade, water between meals.

HIGH PROTEIN MEAT DIET (SUPPLEMENTED).

Vegetables: Irish potatoes, broccoli, swiss chard, lentils, squash, oyster plant, endive, chicory, romaine.

Fruits in Season: Pineapple (canned or fresh), cherries (canned or fresh), peaches (canned).

Berries: Raspberries, blackberries, huckleberries, cranberries, currants, gooseberries.

Melons (in season).

Maple syrup or corn syrup.

Fluids: Coffee (black, one cup daily).

Dressing for salad: *Apple* cider vinegar with sugar. Alternate: Pure olive oil or sesame oil, apple cider vinegar, salt and pepper. Mix salt and pepper with vinegar. To one part of the vinegar mixture add two parts of olive oil or sesame oil. Whip until creamy.

HIGH PROTEIN FIN FISH AND POULTRY DIET.

Foods selected for the trial diagnostic period permit:

Meat: Lamb and mutton (kidneys included).

Poultry: Chicken, turkey and duck. (Livers and giblets frequently.)

Fin Fish: (Particularly herring, salmon, mackerel and sardines in *pure* olive oil.)

Vegetables: Sweet potatoes, raw carrots, beets, artichokes, and alligator pears, brown rice, wild rice, and soy beans.

Fruits in Season: Grapefruit and lemon, lime, dates, figs, dried prunes, dried apricots, pears, plums.

Tapioca.

Fluids: Tea with lemon and sugar, lemonade, water between meals.

HIGH PROTEIN FIN FISH AND POULTRY ALTERNATE (SUPPLEMENT)

Meat, Poultry, Fin Fish: See high protein fin fish and poultry diet.

Vegetables, Fruits and Berries: See supplement of high protein meat diet.

Fluids: Coffee, black, one cup daily.

The use of this diet represents a return to eating simple, basic foods which have been properly prepared. Unfortunately, our modern conception of a proper diet envisions eating a piece of "needled" toast and a cup of coffee for breakfast, a counter sandwich for lunch, and a "chocolate malted" before a salad and ice cream dinner. Attractive, simple, nutritious foods are considered as a radical departure from normal. To induce a large (or small) patient to accept simple foods requires the efforts of a doctor with real sales ability. Unfortunately, foods that are considered "body builders" and "foods that are good for you" are overused today. These same foods by odd fact are also the most frequent "poisoners" of those people who are food sensitive or allergic. So-called nourishing foods are actually forced on our undernourished school children by a mechanized and misguided school dietary program. The undernourishment of children is ofttimes due to forced ingestion of milk which *for them* is a poison. Unfortunately, when any doctor suggests that these personal poisons be removed from the diet, there is an immediate resistance by all parties concerned. If a youngster is forbidden to eat a personally favored food, he may be placed at the head of the resistance list as a most strenuous objector.

Food sensitive youngsters are frequently thin, "pasty faced," nervous, pampered, highly emotional, and hopeless "spoiled brats." They "catch" one cold after another. If your salesmanship is successful and your program is followed, these same rebellious patients will become most agreeable collaborators. In addition to the improvement in personality, they acquire a normal health. Such results will be among your most gratifying experiences.

To summarize statements about food:

1. Select those foods for the diet which are infrequent causes of clinical allergy.

2. All meals must consist largely of the high-protein food, meat. A meat diet will increase energy.

3. The daily vitamin and mineral intake must be adequate.

4. The caloric values can be adjusted to maintain, increase or decrease weight.

5. A variety of food must be properly prepared and appear appetizing.

GENERAL HEALTH MEASURE.

One issue cannot be dodged. Many people are sensitive to tobacco and some are sensitive to alcoholic drinks. Patients who will abstain from liquor and tobacco during the six weeks' trial diagnostic period will do better than those who will not. The relation of alcoholic drinks to allergy is of less importance than tobacco. There is one conclusive test which individuals who may be allergic to tobacco can try. Abstain from all smoking for six weeks and honestly compare your trial period with the previous six weeks' period. Answer yourself honestly regarding the result. Some people can smoke with apparent impunity while others cannot. Cigarettes seem to be the worst offenders. I doubt that anyone would claim an improvement in health because of smoking. Rarely is it possible to benefit by "cutting it down." To get results you must "cut it out."

People afflicted with perennial allergies seem to be those who have insufficient exercise "out-of-doors." Many methods of daily exercise have been tried for urbanites and walking works the best of all. A two-mile daily walk completed before breakfast will do wonders for those patients who live physically inactive lives.

IMMUNIZATION AND DRUGS AFFECTING THE VASOMOTOR MECHANISM.

The methods advocated by Hansel (Hansel, F. K.: Some Experiences with Small Dosage Dust and Pollen Therapy, *Southern Medical Journal*, 38:608, 1945) and also employed by Ashley (Ashley, Rea E.: Dust Therapy in Allergy of the Ear, Nose and Throat, *Annals of Otolaryngology*, 58:417-428, 1949) have been used by me with success. The intradermal skin tests are done with a 1:1,000,000 dilution of Hansel's dust and a 1:1,000,000 dilution of histamine. Following their practice, I also use the skin tests to regulate dosage for the hypersensitive patients rather than as a determination to use or not to use the treatments.

Drugs affecting the vasomotor mechanism have been used with success.

Physical allergy remains as an unsolved problem. Under this heading are grouped those unfortunates known as "draft sensitive" people. They may have suffered some sort of trauma to their heat-regulating mechanism which fails to accommodate for temperature changes. The air-conditioned buildings are unbearable for those people who suffer from physical allergy. Air-conditioning systems using refrigeration cause the most trouble, and those using humidification and dehumidification through a blower are only a little less troublesome. Electric fans will precipitate the usual distressing head symptoms. Changes in temperature when people go from one room to another as well as temperature changes encountered on entering and leaving the water while bathing may cause an onset of symptoms. Histamine immunization plus avoidance is the only hope for these maligned, "fussy" people, as of today.

ELIMINATION OF CONTACTS.

Proper preparation of the living quarters, particularly the bedroom, will eliminate many of the causes of allergy, especially among the inhalants.

The following articles should be removed from living contacts: rugs, carpets, overstuffed furniture, pets, flowers, cosmetics and toilet water.

Pillow and *mattress* must be allergin-free or have allergin-proof covers. *Rooms to be cleansed* with either a vacuum cleaner or with a wet rag. *No cosmetics* are to be used. This includes face cosmetics, powders, toilet water, perfumes and preparations for the hair. There are allergin-free cosmetics available which may be used.

It seems necessary to include some comments on "history taking" combined with tact in the approach to our treatment. All alert doctors agree on the inestimable value of a comprehensive history in making a diagnosis of allergy. History taking is the first, most important, and indispensable step. A history must be thorough and complete. In obtaining a proper history, perseverance and patience plus a sincere effort must be used to obtain the friendly cooperation necessary for the best results. Children, especially, require a leisurely, tranquil, friendly approach by the doctor. The doctor must be calm and maintain self-control at all times. He must disregard the annoyance sometimes caused by parental interference. Once a confidential relationship between the smaller patient and the doctor has been established, it is agreeably surprising to see how complete the frankness and help of your patient can be. This faith and trust on the part of the patient will be most important when you must know of any deviation from dietary rules. All confessions of error must come from the patient, never from the parents in front of the child. This young patient will be deprived of many things he may particularly like, and for him this loss of desired and enjoyed foods will be a serious change in his pleasant living habits. First visits should be used to establish confidence on the part of a child and at the same time convince the parents of the benefits derived from a program which may seem to them like medical radicalism.

Being human, some parents bribe their children with tasty but forbidden foods such as ice cream, chocolate milk drinks, or nut candies. Even grandmother thinks it is smart to buy a grandchild's favor with some of the forbidden foods. Grandma's helpful comments to cover her misdeeds sometimes cause suspicion, and once the child's confidence and faith are lost, they are difficult to regain. Whenever it is possible, the doctor should talk with his little patients alone. He will be more apt to succeed in obtaining full and confidential cooperation. Minor misdemeanors on the part of the patient will then "come out in the wash."

This paper does not deal with symptoms or diagnosis. It is primarily concerned with the treatment of the allergic patient who consults the otolaryngologist. Case histories from my files would strengthen my contentions, but individual case histories are valueless and many such histories are a monotonous repetition of symptoms and methods. The types of cases which have responded in satisfactory numbers are recurrent colds, recurrent and chronic sinusitis, recurrent otitis media and interna (Ménière's syndrome), headaches (including migraine), and the postnasal drip (catarrh). The complete program is presented for your consideration and trial. Thirty years ago the subject of allergy in otolaryngology was ridiculed. Today the modern otolaryngologist recognizes the fact that allergies are important causes of our ear, nose and throat diseases.

COMMENT.

In this paper I have claimed that patients who consult the otolaryngologists because of symptoms due to nonseasonal allergy can be most effectively treated by the otolaryngologist who is conversant with these methods of treatment. The treatments described can cure patients who have suffered from protracted or recurrent sinusitis and render most sinus surgery unnecessary. I have stated that some properly selected patients who have sinusitis require surgery. If such surgery is done by a competent otolaryngologist, who shall at the same time treat the underlying allergy, these patients also can be permanently cured.

I have outlined a program in which I have used a "Dietary Selection Method" for diagnosis and treatment of food allergies.

The need for better living regimes and a correction of an emotionally unstable personality have been given prominence in the treatment of allergies.

The discussion of desensitization has been limited to histamine and a comprehensive dust plus mould combination.

The conditions for which this program has been used successfully by me include the recurrent common cold, recurrent sinusitis, recurrent otitis media and interna, headaches, and postnasal drip.

CONCLUSIONS.

1. Drugs which have an effect on the vasomotor system are of greatest value in the symptomatic treatment of allergies.

2. Increasing numbers of permanent cures are being produced for those patients who have otorhinolaryngologic diseases of allergic origin by proper diet selection, plus the elimination of environmental causes.[*]

3. A combination use of these two methods will produce the maximum in permanent cures for the allergic patient who consults the otorhinolaryngologist.

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[*] Since this talk was delivered in San Francisco, I have found a product which, if it lives up to some of the statements that have been made, will eliminate the necessity for expensive and extensive room changes which are at present necessary in my program. The name of the product is DUST SEAL.[†] *It is a material with which fabrics are treated to eliminate the* damaging dust element.

[†] Arthur F. Coco, M.D., F.A.C.A. (Hon.), Pearl River, N. Y.: "Environmental Excitants of Idioblaptic Allergy (Inhalants)." Reprinted from Annals of Allergy, Volume 6, pp. 501-510, September-October, 1948.

"A CENTURY OF HEALTH PROGRESS" AT QUINCY, ILL., OCT. 13-17, 1950.

The Adams County Medical Society, a component part of the Illinois State Medical Society and the oldest county medical society in Illinois, is doing the unusual by sponsoring a great public celebration to commemorate the centennial of its foundation. The Society was formally organized on March 28, 1850, and has held regular meetings except for one year during the Civil War. The celebration, which will be a nonprofit affair, will be known as "A Century of Health Progress" and will depict the progress of medical science during the past century. The principal features planned are a large technical and educational exhibit hall, a spectacular, colorful pageant under professional direction, a spirited street parade, an educational motion picture theatre. Final plans have not been completed but call for the close cooperation of many of the civic organizations of Quincy in order to assure the success of the project. An estimated attendance of over 60,000 (not including the parade) from northeastern Missouri, southeastern Iowa and central western Illinois is anticipated.

The Society will be grateful for suggestions that will assure the success of the celebration. Dr. Harold Swanberg, W. C. U. Building, Quincy, Ill., is serving as general chairman, Adams County Medical Society Centennial Celebration.

DETERMINING FACTORS IN COMPOSING AND ANALYZING SPEECH-HEARING TESTS.*†

Part I: The Influence of Frequency Composition of the Speech Sounds on the Articulation Curves as Related to Pattern of Hearing Loss.

WILLIS C. BEASLEY, Ph.D.,‡
Bethesda, Md.,
and
HARRY ROSENWASSER, M.D.,
New York, N. Y.

In recent years renewed attention has been given to testing and appraising the hearing function by means of one or another form of speech-hearing test. The voluminous literature that has evolved attests to this awakened interest. Testing the hearing by means of the live voice was the first type of hearing test, and the interest and basic appreciation of the value of this kind of test was only temporarily interrupted by a rather enthusiastic swing to pure tone audiometry. For the clinician as well as for those interested in the purest form of physiological acoustics, for the educator as well as for those concerned with the applied aspects of aural rehabilitation by means of hearing aids, there should be no conflict or competition between the two methods of studying the hearing function.

Sensing the presence of pure tones at faint levels of loudness is not the same psychophysiological function as perceiving patterns of speech sounds of whatever composition at the various tolerable ranges of loudness and degrees of clarity of which the normal or pathological organism is capable.

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., New York, N. Y., Jan. 5, 1950.

†A study from the Department of Otolaryngology, Mt. Sinai Hospital, New York, N. Y.

‡Biophysicist, U. S. Public Health Service.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 7, 1950.

Pure tone audiometry is a sensitive probe of the delicate functioning of the hearing mechanism, and has a permanent and indispensable place in both the clinic and the laboratory; but it does not *measure* the ability to perceive human speech patterns, nor can one predict with certainty exactly how well an individual can perceive speech sounds from an inspection of the pure tone audiogram. If one is interested in the basic physiological condition of the hearing mechanism there is no better means of exploration than with pure tone audiometry with its vast complement of variations. If one is interested in the *perceptual capacities* of the hearing function, the most direct answer can be found by means of speech-hearing tests.

Extensive study of the various forms of speech-hearing tests has been made and reported during the past eight to 10 years. Electroacoustics has evolved to the point where the techniques of recording speech and controlling the performance of reproducers are rather common knowledge and excellent equipment for such purposes can be assembled rather readily. The time is here for developing standards in the field of speech-hearing tests. Physical standardization of equipment is no problem, but there remains considerable indecision as to the content of the speech-hearing tests themselves. The results from our study, using a unique type of speech-hearing test, are particularly timely, because they indicate quite clearly that distinctly different results can be obtained when due consideration is given to the frequency composition of the test words.

II. DATA AND EQUIPMENT.

The data upon which this report is based were drawn from tests that were given routinely to some 850 patients at the Ear, Nose and Throat Clinic of the Mt. Sinai Hospital, New York, during the period from March, 1947, through November, 1949. Approximately 200 of these patients had no significant impairment of hearing, hence their data were used as a normal reference in making comparisons with the data collected for the others who had varying degrees and patterns of hearing loss. In addition, a special prolonged series of tests were given to a selected group of the subjects with normal hearing, as judged from pure tone audiograms.

Both kinds of tests were given in a former treatment room, approximately 18 feet by 20 feet by 12 feet (ceiling), which was acoustically treated but not soundproofed by any means. A window-type air conditioner helped to maintain comfortable temperature during hot days, but this machine was necessarily cut off during the conduct of tests. The ambient noise level in the room under typical testing conditions ranged from 50 to 56 db. The acoustic seal provided by the earphone caps was definitely beneficial, therefore, because of this relatively high ambient noise level.

All patients included in this analysis were given complete pure tone audiometer tests by air and bone conduction with a standardized Maico Model E-1 Audiometer. Most of the air conduction audiograms included all of the 11 frequencies available on the audiometer, extending from 125 to 10,000 cycles. Bone conduction tests were made at each of the eight frequencies ranging from 125 to 4,000 cycles. A specially constructed and calibrated white noise generator was used for masking, when indicated.

In addition to the audiometer tests, all patients were given a new type of speech-hearing test (described later) using an Acousticon Model A-1 speech audiometer as the standardized reproducer. This instrument provides the means for controlling the transmission of test materials from either recordings or live voice sources to the listener by way of earphones (air conduction), free field (loudspeaker) or bone vibrators. Suitable controls on the instrument panel permit testing either ear separately or both ears simultaneously by air conduction. In the present tests, the ANBH-1 type earphones (double headset) with ear caps that provide a good acoustic seal were used throughout.

A monitoring meter and electroacoustic level control on the panel permits maintaining the acoustic output of the reproducers at predetermined calibration levels. A standard attenuator with a range of 50 db in steps of 5 db, with an auxiliary 55 db attenuation pad, provides a useful range of 105 db for the sound pressure level of the speech sounds. For all of the speech-hearing tests in this study the speech sounds were reproduced from standardized recordings that were engineered at the Acousticon Laboratory. The master recording was made by the National Broadcasting Company, Inc., New York, by their standard orthacoustic method, and the printings were made on 10-inch laminated shellac discs. These records are played at 33-1/3 r.p.m., have a standard groove, and are played back with a 3 mm. stylus. The recording includes a 1,000 cycle test tone which is used for calibration purposes. A light weight pick-up arm and high quality variable reluctance cartridge with a permanent sapphire needle were used during the period of these tests.

The method of calibration which was in effect throughout our period of testing involved relating a value on the monitoring meter of the instrument to the sound pressure levels produced on a standard 6 cc. coupler designed for use with the ANBH-1 earphone. The procedure used for standard calibration was to set the amplifier gain so that the 1,000 cycle test frequency produced a steady sound pressure level of 130 db above 0.0002 dynes/cm^2 with the earphone in place on the 6 cc. coupler when the panel attenuator was set to give maximum output. The gain setting required was noted on the panel monitoring meter. This reading was used in the course of testing to maintain the acoustic output of the earphone at the required level. In addition to this reference calibration, readings were taken also with a standard V. U. meter connected across the output of an artificial ear to measure the sound pressure level in volume units (V. U.) delivered by one receiver on the 6 cc. coupler for each word employed in the speech-hearing test. These V. U. meter readings give a measure of the effective maximum sound pressure level of each test word. Since the words employed in this test are all of the consonant-vowel-consonant type, the V. U. readings are very nearly measures of the effective maximum sound pressure level of the *vowel* component of each word. The consonant components of each word range from about 5 to 32 db less than the interposed vowels. On this particular recording, the V. U. readings for individual words range from 121 to 129 db. Most of them concentrate in the range from 123 to 127 db. In plotting the results of the speech-hearing tests on the charts included in this report, the average V. U. readings for the individual test words was used as the basic reference.

III. BEASLEY'S DIFFERENTIAL SPEECH-HEARING TEST.

The unique feature of this speech-hearing test is the critical frequency composition of the individual words and the manner of presenting and scoring the test material. All other speech-hearing tests of which the authors have knowledge include in any scored set as presented in a test an almost complete assortment of the vowel and consonant sounds that occur in a "randomized" order as the test proceeds. Such tests, when used with a standardized and calibrated instrument for reproducton, measure an individual's overall ability to perceive isolated words generally. The idea for a different type of speech-hearing test was worked out by Beasley during the period from 1939 to 1941, primarily as a result from the analysis of data collected during his nationwide study of deafness in the population in connection with the National Health Survey.[1,2]

In devising this test, immediate use was made initially of the detailed data given by Fletcher[3] on the energy *vs.* frequency composition of consonants and vowels and the influence on articulation ratings for the various individual sounds resulting from introducing high pass and low pass filters at various cutoffs into the transmission circuit. A detailed table was set up for all of the speech sounds and a rating scheme devised for selecting the most differentiating phonetic elements of speech sounds. As an entirely empirical result from this analysis, it was concluded that the energy-frequency spectrum can be differentiated in relation to the largest number of individual speech sounds in the region of 1,500 cycles. On this basis vowels and consonants were selected from the table to meet the following requirements:

1. Those that show the greatest reduction in articulation when only frequencies above 1,500 cycles are eliminated and are influenced the least when only frequencies below 1,500 cycles are reduced or eliminated. These were classified as high frequency speech sounds.

2. Those that show the greatest reduction in articulation when only frequencies below 1,500 cycles are eliminated and are influenced the least when only frequencies above 1,500 cycles are eliminated. These were classified as low frequency speech sounds.

From these vowels and consonants a list of real words of the consonant-vowel-consonant type was composed. It should be noted that *all three units* in each word were selected and combined to give frequency discrimination. The resulting test was organized into lists of high frequency words (HF) and low frequency words (LF). The two different lists are scored separately and the two ratings of articulation result. One set of words, which comprised the materials of the test as used in our present study, is shown in Fig. 1. This is an actual section from the score sheet as used during the testing, and the word lists are arranged in the order as they appear on the first half of the standardized recording. These first six lists of words are repeated on the second half of the record, but in shuffled order to reduce the tendency of memorizing. The whole recording presents altogether 12 lists of 10 words each.

	db						
		hf-1	lf-2	hf-3	lf-4	hf-5	lf-6
*bone	*masked--db___	p ea ch	l ai r	sp a ce	h ai r	p ea ce	l o re
		t ee th	b o re	ch a t	m o re	f a t	m u m
		ch a se	h u m	f ee t	b u m	sh a pe	r oa r
*air		p a t	w a r	t ea ch	m a r	p i tch	w a ll
		f i fth	r o be	s a fe	r oa m	ch ea p	m o b
		s ea l	m a re	f ea st	w e ll	t a ste	r u b
*left		f a st	b e ll	ch a fe	h o le	s a f	m oo n
		ch ie f	h o me	c ea se	r a re	f a ce	w ea r
		p a ste	r o b	p a ss	b o b	ch ea t	r u m
		f i st	b a ll	th ie f	h a ll	s ee p	h u ll
*right	C						
	V						
	S						

Fig. 1. Basic lists of high frequency words (hf) and low frequency words (lf) in Beasley's differential speech-hearing test as used in the present study.

The words are listed alternately high frequency and low frequency words as indicated at the top of each column. For convenience in scoring, the phonetic elements in each word are separated by a space. The technician conducting the test strikes out the unit or units in the word as they are missed, and can quickly score the column before passing to the next list. This is a definite advantage in clinical testing where time is a critical consideration. At the foot of each column are blank spaces for writing the consonant, vowel and word scores and percentages. In the present study, only consonant scores have been used as the basis for plotting articulation curves, the reason being that the consonant sounds give the most critical differentiation between high frequency and low frequency perception. Suitable spaces are blocked off on the form for indicating which ear was tested, whether by air or bone conduction, and the attenuator setting in decibels at the top of each column. The order in which different intensity levels are used may be varied at will, and likewise the lists that are played at particular levels may be varied at will. This is a definite advantage in clinical testing.

This form of speech-hearing test was subjected to considerable engineering standardization at the Acousticon Laboratory in order to obtain a high quality recording and a standardized method of control when the records are used with the speech-audiometer.

IV. METHOD OF CONDUCTING THE TESTS.

As a rule, each patient was given the pure tone audiometer test first, followed by the speech audiometer test. Frequently, a patient was given both tests at the same visit. If not, the second test followed in a few days.

In taking the speech audiometer test, the subject listens to the words through the earphones, one ear at a time, and pronounces the word as heard. The technician listens to the subject call off the words, and strikes out the unit or units of each word incorrectly called, or checks out the whole word as correct, if that is the case. There is sufficient time between each list of words for the technician to score each column and write down the number of consonants, vowels and words called correctly. This is a definite aid in determining the manner of conducting the test on a particular patient.

A typical procedure in giving this test is to play the first two lists—one HF and one LF—at a sound pressure level of 80 db, then to work up or down in level, depending upon the response of the patient. The objective in each test is to determine the maximum level at which the patient obtains a zero score and at least two levels 10 db apart at which the maximum scores are obtained. Between these two end-points, measurements are made at 10 db intervals. When the level of scores drops abruptly in a particular 10 db step, another test is conducted at the 5 db interval. As the percentage scores approach zero, 5 db intervals are employed. For a typical clinical run, it is usually sufficient to employ only six different levels, if these are chosen judiciously to include the two end-points, which are essential. It is often necessary to repeat the levels used at the beginning, especially when subjects are taking the test for the first time, for usually some learning or adaptation to the observations is required.

V. RESULTS OF TESTS ON SPECIAL NORMAL GROUP.

In order to have a satisfactory normal reference against which the data collected on clinical patients could be compared, a series of tests was given to a selected group of normal subjects. These 50 normal subjects were drawn largely from existing files, and the group consisted entirely of persons with normal pure tone audiograms for both ears regardless of age and sex. There were no appreciable losses for any tones, even at the higher frequencies. Each subject was given an extended series of speech audiometer tests in addition to a complete pure tone audiometer test before and after the speech audiometer tests.

The series of speech audiometer tests extended over a period of six months. Each subject was tested on each ear five times. The tests were divided between three different days for each subject at intervals ranging from a few days to a month. In each test run for either ear, one HF and one LF list was

results of speech audiometer tests and differential ratios between relative amounts of hearing loss for air and bone conduction.

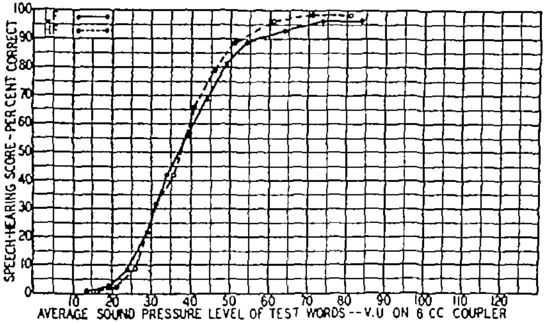

Fig. 3. Articulation curves for high frequency words (dashed curve) and low frequency words (solid curve) resulting from the prolonged series of tests given to 50 selected normal subjects.

The dashed lines bordering either shaded area on this chart indicate the limits of the semi-interquartile range, which includes the middle 50 per cent of the measurements at each audiofrequency. The heavy black line in the middle of each area indicates the median value (midpoint of frequency) for each distribution. We have used this median value for the normal group as the actual zero reference for normal hearing by air conduction in the charts shown in Figs. 4, 6, 8, 10 and 12. The irregularities of this median curve in Fig. 2 reflect largely the basic calibration characteristics of the pure tone audiometer used. By using the experimental "normal" curve as the zero reference, considerable smoothing of the curves in the charts referred to is accomplished, and facilitates interpretation of the relative differences beween groups in the ensuing analysis. For the sake of complete accuracy, the numerical values of the points on this normal median curve are given here. These values were subtracted from the experimental median values for the patient data before plotting on the five charts referred to above. The original curves can be restored by anyone interested in doing so simply by adding these reference values to the points on the curves as plotted.

TABLE 1. NUMERICAL VALUES OF THE MEDIAN CURVE FOR 50 NORMAL SUBJECTS, AIR CONDUCTION.

Audiofrequency (Cycles)	Value Db on Chart
125	19.4
250	14.9
500	11.4
1,000	8.2
1,500	11.4
2,000	3.9
3,000	11.3
4,000	— 0.1
6,000	10.5
8,000	3.6
10,000	11.7

The articulation curves for LF and HF consonants resulting from the extended series of speech audiometer tests given to the 50 normal subjects are shown in Fig. 3. Each point on each curve represents the scoring of 10,000 consonants. There were 120,000 consonants scored for each curve, or a total of 240,000 experimental observations for the two curves. They represent the normal reference for the speech audiometer tests as conducted under our conditions as described. It is noted that the curves for HF and LF words are not significantly different for the normal subjects. A composite average between the two curves does not differ significantly from the normal articulation curves for PB word lists as reported by Davis[4] and Thurlow.[5] This suggests that equipment and techniques for standardizing speech hearing audiometers for general use are well within the realm of practicality. An unexpected finding is that even though the scoring is based entirely upon perception of consonants, the articulation curves are nearly equal for normal subjects. It should be pointed out in this connection that the *low frequency consonants* used in this test are on the average from 8 to 20 db higher in peak values than the *high frequency consonants*, even though the V. U. sound pressure levels of the interposed *vowels* do not differ more than 2 db on the average for the HF and LF lists.*

VI. ANALYSIS OF DATA FOR CLINICAL PATIENTS.

The body of records collected on the 850 patients provides valuable information which may be analyzed in a variety of ways to reveal fundamental relationships between pure tone audiometric tests, by both air and bone conduction, and the speech audiometer tests. The present analysis aims at one specific objective, namely, to determine whether the differential speech audiometer test used in this study serves to reveal any useful information that cannot be obtained readily by using other currently available test materials. The organization of data in the subsequent analysis was designed specifically to test the theoretical assumptions from which the differential speech audiometer test originated.

The first step was to derive an index figure for the *degree* of pure tone hearing loss by air conduction for either ear of each patient. For this index, a simple average was computed for the hearing losses at 1,000, 1,500 and 2,000 cycles. This is designated as the middle range loss (MRL). This average was computed for each individual pure tone audiogram, either ear, and written on the patient's statistical chart.

The second step was to compute a weighted average for the frequencies above 2,000 cycles. The weights assigned were: 3,000 cycles, one-third; 4,000 cycles, one-third; and the average loss for 6,000, 8,000 and 10,000 cycles, one-third. This weighted average is referred to as the high frequency loss (HFL). This average likewise was computed and written on each patient's statistical chart.

A code was entered for either ear on each chart next in accordance with the following groupings:

Group 1: MRL, 15 db or less.

Group 2: MRL, 16 to 30 db.

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*The S. P. L. values for the consonants were determined from continuous film records of cathode ray oscilloscope projections of the speech patterns.

Group 3: MRL, 31 to 45 db.
Group 4: MRL, 46 to 60 db.
Group 5: MRL, 61 to 80 db.
Group 6: MRL, 81 db and over.

A code was entered then on each chart for either ear to specify one of three relationships between the relative values of MRL and HFL, as follows:

Subgroup A: HFL equal to or less than MRL.
Subgroup B: HFL from 1 to 10 db greater than MRL.
Subgroup C: HFL is 11 db or more greater than MRL.

The frequency distributions were then compiled for all of the original pure tone audiometer tests for all individual ears classified according to the above groups. While sorting the data sheets, we noted that there were only a few instances in which ears classified as 5C and all of Group 6 attained any perception at all on the speech audiometer test. Consequently, these groups were excluded as not serving any purpose in the present analysis. Statistical constants were computed for the frequency distributions of audiometric measurements for each grouping. Study of these numerous tables indicated that the trends could be represented suitably by the median curves of the hearing loss data for each subgroup and depict the relationships existing. These median curves are plotted in the charts of Figs. 4, 6, 8, 10 and 12.

Similarly, the articulation curves for LF and HF consonants were computed for each of the subgroups. These are plotted in the charts of Figs. 5, 7, 9, 11 and 13.

Each subgrouping yielded a sufficient sample to be useful. The number of ears which became classified according to the preceding definitions are given in Table 2.

A careful and detailed examination of the pure tone audiometric and speech audiometer charts, making comparisons between each associated pair, then successive comparisons between the charts for different groups, will enable the interested reader to derive a number of meaningful observations.

TABLE 3. DISTRIBUTION OF EARS ACCORDING TO CLASSIFICATIONS DEFINED IN THE TEXT.

Major Group	Subgroup A	B	C	Totals
1	264	264	61	589
2	75	106	77	258
3	70	80	60	210
4	69	66	34	169
5	54	39	—*	93
Totals	432	555	232	1,319

*Excluded.

We shall call attention to only the major observations that are of particular interest in relation to the differentiations revealed by differences between the articulation curves for LF and HF words and the relationship beween these differences and the character of the median audiograms for the correponding subgroups.

As the first example, consider the charts for Group 1 in Figs. 4 and 5. The most indicative observations to note for this paired comparison are that the median audiogram curves for Groups 1A, 1B and 1C for the range from 125 to 1,500 cycles are quite uniformly horizontal lines and fall within a range of 5 db; whereas, in the range above 1,500 cycles, the 1A is continuous with that portion for the LF range, the 1B curve *shows a drop of 15 db in the HF range, and the 1C* curve shows a sloping drop in the HF range averaging about 30 db. On the corresponding speech-hearing articulation curves for this group, as illustrated in Fig. 5, the LF articulation curves (solid lines) are close together, have very nearly the same curvature, and are close to the normal curve reference. In contrast, only the 1A articulation curve for HF words (dashed line) remains closely associated with its LF mate on the graph; the 1B HF curve departs moderately from its LF mate, but the 1C HF curve departs conspicuously from its LF mate. These relative differences between each pair of articulation curves follow almost exactly the characteristic differences between the median audiograms of Group 1.

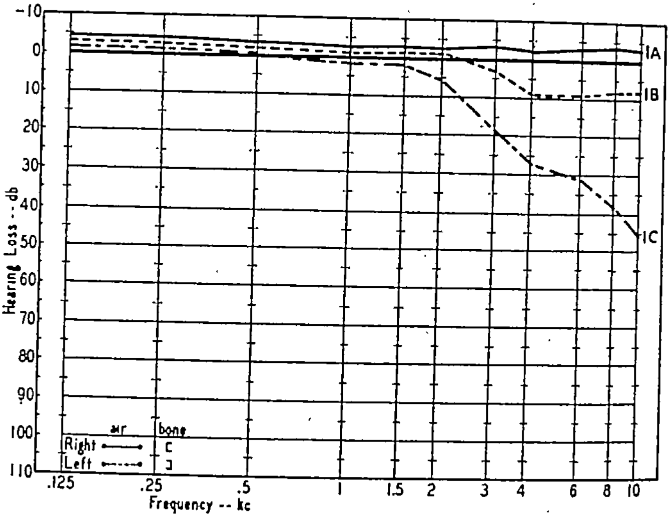

Fig. 4. Median air conduction audiograms for Groups 1A, 1B and 1C. The zero reference line in this figure, as well as in Figs. 6, 8, 10 and 12, is based upon the median values for the selected normal groups, air conduction, Fig. 2.

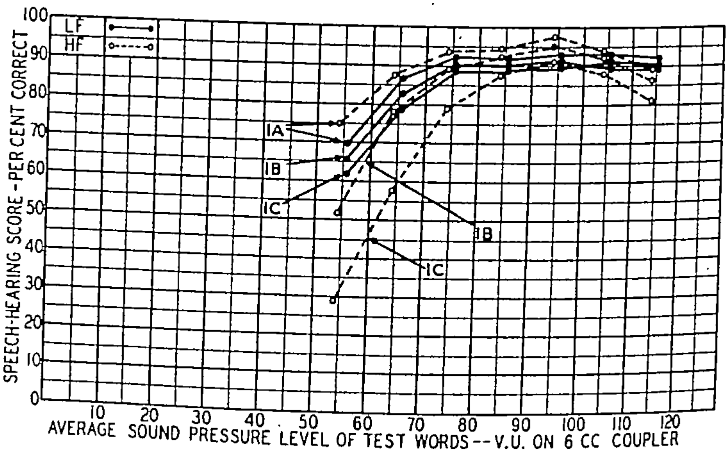

Fig. 5. Articulation curves resulting from differential speech-hearing tests given to Groups 1A, 1B and 1C. Compare with Fig. 4.

Similarly, examine the pair of charts for Group 2 in Figs. 6 and 7. Here again the portion of the median audiograms for the range from 125 to 1,500 cycles are close together for

Subgroups 2A, 2B and 2C, whereas the differences between the portion for the HF range are separated more conspicuously. Note that the order of the LF and HF portions of the median audiograms are reversed, even though slightly; that is, the 2C LF portion shows the least amount of hearing loss,

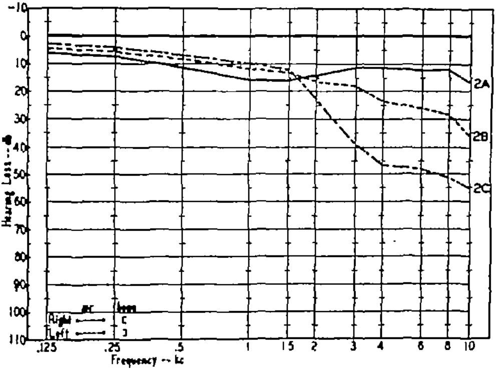

Fig. 6. Median air conduction audiograms for Groups 2A, 2B and 2C. Compare with Fig. 7.

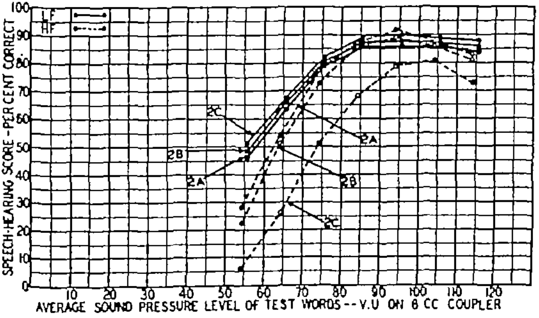

Fig. 7. Articulation curves resulting from differential speech-hearing tests given to Groups 2A, 2B and 2C. Compare with Fig. 6.

whereas the 2C HF portion shows relatively the greatest amount of hearing loss. On the HF and LF articulation curves for these groups, as shown in Fig. 7, the LF curves are close together and show less impairment for LF words than the HF curves show for HF words. The latter curves deviate from the LF curves conspicuously. In particular, the 2C curve is widely displaced relative to the 2A and 2B HF curves as well as in relation to all three of the LF curves. This is the order and degree of differentiation exhibited by the median audiograms.

The differential trends revealed by a paired comparison of the charts for Group 3, as shown in Figs. 8 and 9, exhibit a particularly unique reversal of relationships. Note that the order of the LF portion of the median audiograms (see Fig. 8) are conspicuously reversed, that is, the average hearing loss for Group 3C is about 15 db *less* than that for Group 3A in the low frequency range; whereas, the average hearing loss for Group 3C is about 30 db *greater* than that for Group 3A in the high frequency range. The order of the articulation

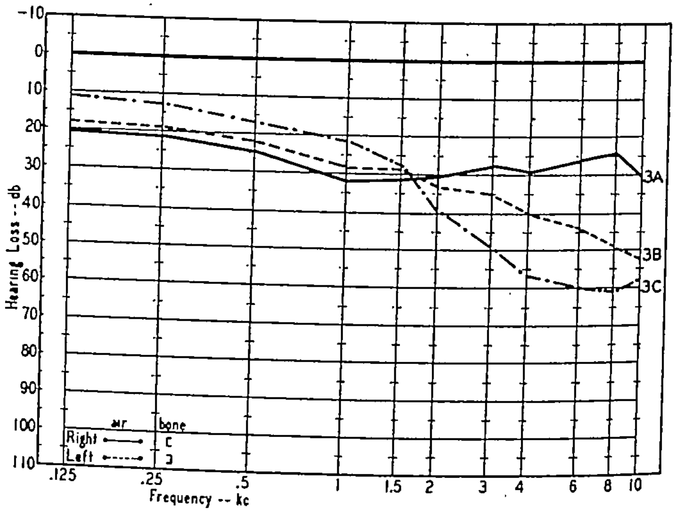

Fig. 8. Median air conduction audiograms for Groups 3A, 3B and 3C. Compare with Fig. 9.

curves for LF and HF words is reversed as shown in Fig. 9, corresponding to the reversal shown by the median audiograms; moreover, the displacements of the articulation curves correspond proportionately to the relative differences in degrees of hearing loss revealed by the median audiogram curves.

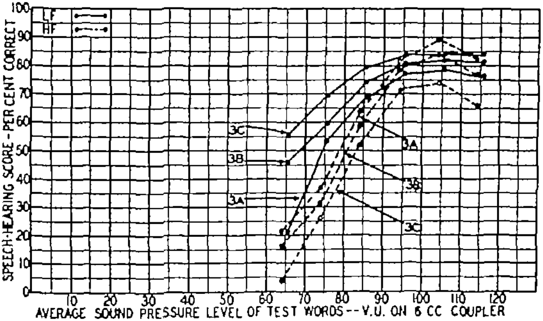

Fig. 9. Articulation curves resulting from differential speech-hearing tests given to Groups 3A, 3B and 3C. Compare with Fig. 8.

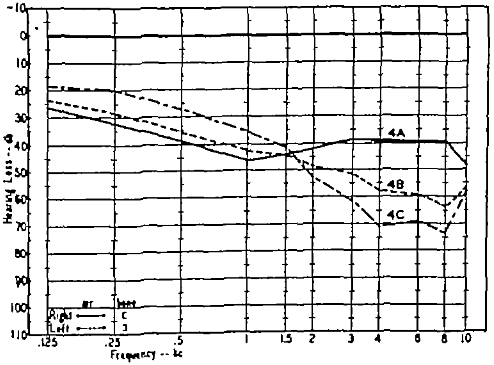

Fig. 10. Median air conduction audiograms for Groups 4A, 4B and 4C. Compare with Fig. 11.

Similar reversals and character of trends as described for Groups 3 and 4 are exhibited for Group 4, Figs. 10 and 11, and for the two subgroups of Group 5, as shown in Figs. 12 and 13.

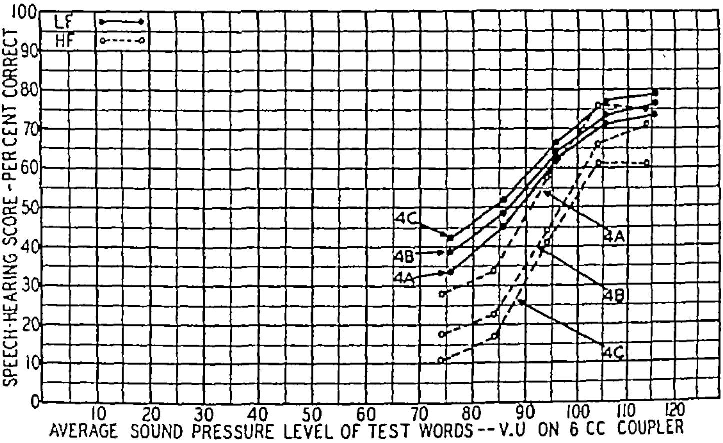

Fig. 11. Articulation curves resulting from differential speech-hearing tests given to Groups 4A, 4B and 4C. Compare with Fig. 10.

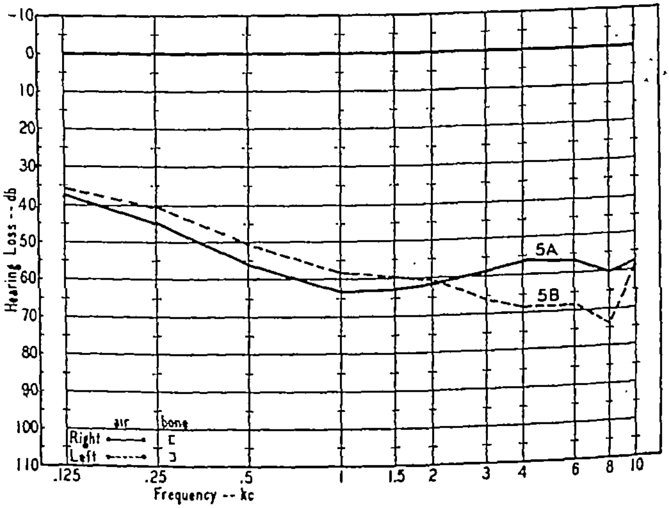

Fig. 12. Median air conduction audiograms for Groups 5A and 5B. Compare with Fig. 13.

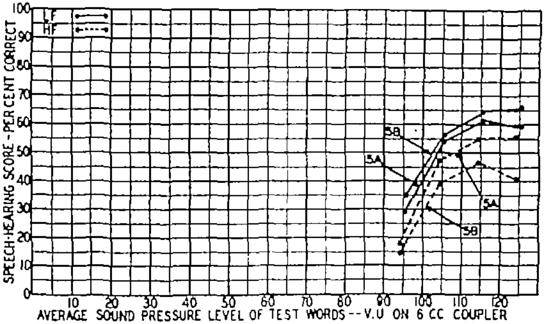

Fig. 13. Articulation curves resulting from differential speech-hearing tests given to Groups 5A and 5B. Compare with Fig. 12.

VII. SUMMARY.

1. A new kind of speech-hearing test, known as the Beasley Differential Speech-Hearing Test, is described. This test is composed of monosyllabic words of the consonant-vowel-consonant type, which are divided into two lists on the basis of the energy-frequency characteristics of the phonetic elements. The words in the two lists are designated as low frequency (LF) and high frequency (HF) words. The initial and final consonants in these words contribute more than the vowels to the differentiating efficiency of the test and, hence, scoring is based upon perception of consonants. The test is organized and administered in such manner that two articulation curves result: one for LF and one for HF phonetic elements.

2. This test was given to 850 patients, of whom about 500 had hearing impairments ranging from slight to very severe. In addition, pure tone audiograms by air and bone conduction were obtained routinely on each patient. A prolonged series of tests were given to 50 normal subjects as a control for comparison with the patients' data.

3. In this report, the data were analyzed by a group trend method to determine whether the HF and LF articulation curves bear any consistent relationship to the pattern of pure tone audiograms. The results, as depicted in 12 charts based upon statistical calculations for grouped data, indicate consisently that:

   A. For the subjects having normal pure tone audiograms, there is no significant difference between the positions and curvature of the LF and HF articulation curves.

   B. For subjects with impaired hearing, the displacement of the LF and HF articulation curves from the position of the articulation curve for normal subjects is approximately proportional (but not equal) to the average amount of hearing loss for pure tones at 1,000, 1,500 and 2,000 cycles.

   C. For subjects with different *patterns* of pure tone audiograms, the LF and HF articulation curves are displaced relative to each other in amounts and direction that are proportional to the relative average hearing loss for pure tones below 1,500 cycles (LF) and above this reference (HF).

4. Although these trends, which are based upon grouped data, are consistent throughout, there is a significant number of cases (approximately 28 per cent) in our records that do not conform. Prediction of speech-hearing ability from the pattern of pure-tone audiograms is not valid for individual cases. The authors expect to explore the reasons for these exceptions to the basic group trends in a subsequent analysis.

5. The demonstrated efficiency of this particular speech-hearing test in revealing differences between the abilities to perceive low frequency and high frequency phonetic elements of speech-sounds has a practical significance for the otologist, especially in evaluating end-results after the fenestration operation for clinical otosclerosis.

6. Similarly, this differential speech-hearing test has a direct application to the selection of hearing aids in individual cases, and appraising the *nature* of hearing improvement accomplished by different hearing aid fittings.

ACKNOWLEDGMENT

The authors acknowledge with appreciation the cooperation of the Engineering Department of the Acousticon Laboratory in providing equipment and technical services in support of this study.

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THE RECRUITMENT OF LOUDNESS PHENOMENON.*†

EDMUND PRINCE FOWLER, M.D.,
New York, N. Y.

The "recruitment phenomenon," after twenty-odd years, although universally accepted, is still not clear to many otologists; they do not understand the loudness function of the ear. It is for these reasons and also to keep you informed of the newer tests and uses of "recruitment" that this brief paper has been written.

To orient ourselves let us consider just what is meant by "recruitment." When a sound (a pure tone, a band of frequencies, a noise, speech, or any sound) is gradually increased in intensity above the threshold of a normal hearing ear, or an ear blocked by a purely impedance lesion, it will be sensed with an orderly logarithmic increase in its loudness. In other words, the loudness function in such ears has a fixed value. It varies somewhat with frequency.

If one ear has normal hearing and the opposite ear an impedance deafness, the threshold of the deafened ear will be a definite number of decibels higher than the normal threshold. If the sound is increased 10, 20, 30 or any number of decibels over threshold in each ear the difference in decibels at threshold will be maintained at the higher intensity levels. The deafened ear will at all intensities of sound hear the sound the same number of decibels fainter than the normal ear, as indicated by the threshold determinations and will require the addition of its threshold loss in decibels to bring it up to the same sensation of loudness as the opposite better ear.

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 25, 1950.

†This paper in part is a condensed version of a paper read before The International Congress of Otolaryngology in London, July 19, 1949.

Editors' Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

On the other hand, if one ear has normal hearing, and the opposite ear is deafened by a neural or so-called "nerve deafness," the difference between the two ears at threshold will disappear as the sound is increased over threshold, until at high intensities the sound appears just as loud (and sometimes even louder) to the deafened ear as it does to the normal ear. This pathological or pathophysiological increase in the increment of loudness is called "recruitment of loudness," and for short, "recruitment." It is pathognomic of auditory neural lesions.

Once recruitment is understood it is apparent that it may be demonstrated in many ways. I shall describe six techniques which are usable as tests for its presence.

Using a single receiver monaurally transferred from ear to ear, or preferably twin receivers, switch the sound from the audiometer alternately into the right and left ears. By trial and error balance the loudness of the testing frequency binaurally. This method of procedure is called the "alternate binaural loudness balance" technique.

The recruitment phenomenon was discovered and measured by this method some 25 years ago. I first reported it in 1928. It may be most dramatically demonstrated in monaural pure neural deafness when the hearing in the opposite ear is normal. We commonly use the term "nerve deafness" as synonymous with a deafness caused by lesions anywhere in the neural mechanism of hearing. It is more accurate to use the term "neural deafness" and qualify it as "peripheral, central and cortical" when we wish to differentiate lesions in the different levels of the auditory neural pathways.

Fig. 1 shows the audiograms of the right and left ears of a patient with monaural pure neural deafness at 2,000 c.p.s. and above. The left ear shows a loss of 60 db and the right ear no loss. Now, although at 2,000 there is a difference of 60 db in the thresholds of the right and left ears, a very loud sound (in this instance 90 db above normal threshold) appeared to the patient to be equally loud in the normal and the severely deafened ear. In other words, the number of decibels required to binaurally balance the sound was the same.

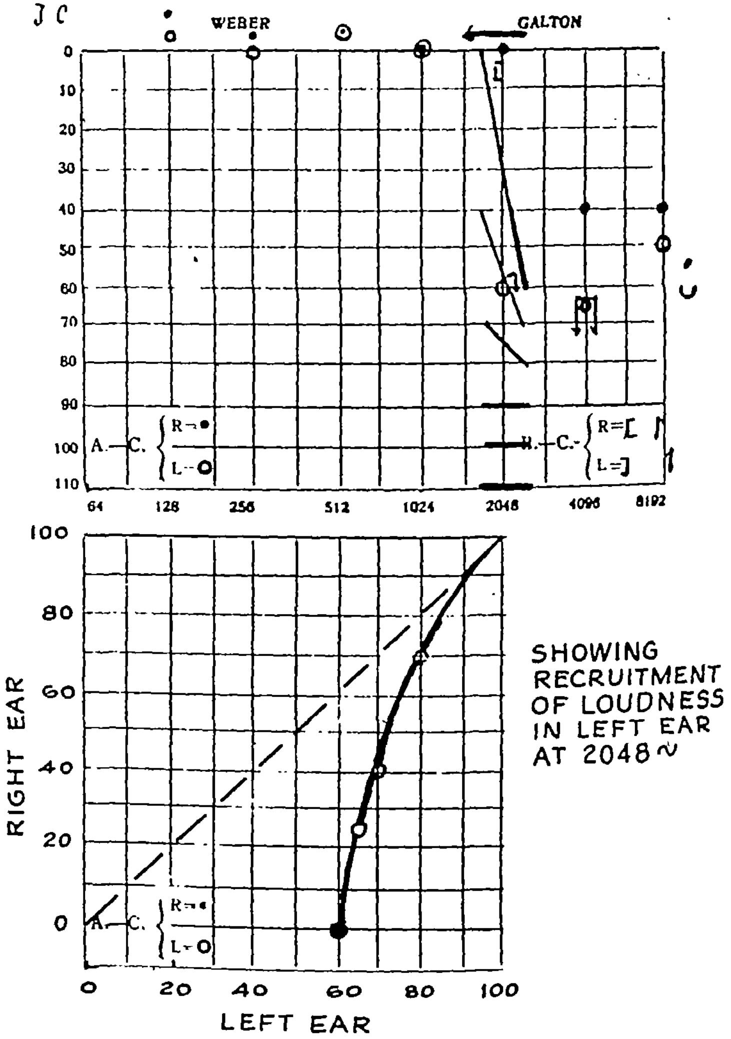


Fig. 1.

In the standard audiogram chart the pathological threshold (the decibel loss) is plotted below the average normal threshold line. It is upside down insofar as above and below threshold is concerned.

If we plot loudness in the right ear to the left of the frequency ordinate, and the loudness necessary to balance it in the left ear to the right of the ordinate, a line drawn between these points will show by its length, its slope and the decibel intensity indicated at each of its ends, the intensities necessary for equal loudness in the two ears. A series of lines joining all the equal loudness plottings will produce graphically what may be called a pole ladder, or a phantom ladder graph (a ladder with rungs but without sides).

The equal loudness balance line joining the thresholds in this graph shows that there is here a difference in the intensities of 60 db at 2,000 c.p.s. Increase the intensity of the sound in the deafened ear to 10 db above its threshold—bringing it to 70 db above normal threshold—now apply the same sound to the right ear, and by trial and error adjust it until it equals in loudness the 70 db sound as heard in the left ear. As illustrated in the audiogram, the right and left ears balance in loudness at 40 db and 70 db, respectively. The intensities necessary to obtain this balance show a difference of 30 db, whereas at threshold there is a difference of 60 db. There has been an increase in the sensation of loudness (an increment over what might have been expected) of 30 db in the left ear. Now increase the intensity in the left ear another 10 db, making a total of 80 db above normal threshold. The right ear now requires 70 db over its threshold to balance this 80 db sound in the left ear. The ears are now only 10 db apart in their equal loudness plottings. (The left ear has now recruited 50 db of loudness.) Increase the left ear to 90 db and the intensity necessary for equal balance in the right ear is now also 90 db. In some way the left ear has recruited an added increment of loudness sufficient to completely overcome the difference of 60 db shown at threshold.

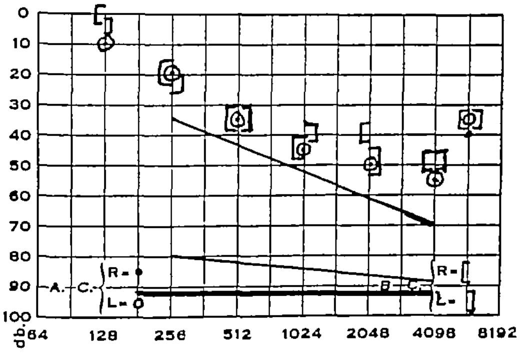
The phenomenon is shown plotted in another way in the graph below the chart. The intensities heard by the better (right) ear are measured along the ordinate and those used to balance them in the poorer (left) ear along the horizontal base line (the abscissa). In this instance only the 2,000 frequency is illustrated. The 45 degree line represents the equal loudness balance plottings of two ears with normal loudness function. The curved line joining the equal loudness plottings of the normal right and deafened left ears shows the amount, degree and changing increment of the recruitment of loudness in the left ear.

Recruitment occurs only in the very limited or wide frequency bands affected by neural deafness of any degree, and at any and all frequencies. It was never found in an ear with pure obstructive (impedance) deafness, or with the shadow curve of monaural total deafness. In bilaterally equal neural deafness, because under such circumstances both ears recruit equally, recruitment is present although not apparent when using the equal binaural loudness balance test; however, it is present in such cases in both ears and may be detected by other techniques.

Notice that the greatest recruitment occurs just over threshold and that its increment gradually diminishes with increasing intensities of the testing sound.

The initial increase of recruitment is often startling. For instance, it may require a sound 25 or more decibels over threshold in a near normal ear to equal in loudness one 5 db over the threshold in a severely neurally deafened ear. It is this observation which supplies us with a simple test for detecting the recruitment in instances where both ears have similar losses of heaing or in which there is only one ear to test, the other being totally deaf. To detect this initial (just above threshold) over increment in loudness, us a 1 to 2 db increase over the threshold intensity. Even 2½ db is usually satisfactory for routine clinical use. If the patient is easily and quickly cognizant of a 1, 2 or 2½ db increase in intensity over threshold it indicates the presence of the recruitment phenomenon. In a normal ear or an ear with impedance lesions, not only is the threshold indefinite but an increase of 2 db over threshold is not noticed, or is detected with difficulty, particularly by untrained observers.

The reason why the threshold and a slight increase over threshold are so definite in neural deafness is because of "recruitment." The sound, or increase, appears suddenly, and, therefore, there is no doubt as to whether it is or is not heard, or as to whether the increase is or is not heard. At a fraction of a decibel below threshold it is not heard; at a fraction decibel increase it is heard, and a 1 or 2 db increase may sound like a 5 or 10 db increase. The threshold becomes more and more indefinite the greater the coincidental obstructive lesions. The reason is that the greater the impedance lesion the less proportionally is the neural deafness, and the less the neural deafness the less the initial recruitment. If recruitment ceases at any level below saturation intensities it indicates that there is a coincidental impedance deafness.



UNILATERAL AND BILATERAL BIFREQUENCY BALANCE BY A.C.

FIG. 2.

If the threshold curves of either one or both of two ears are not horizontal, you may balance a high tone, say 2,000 or 4,000 against a lower tone, say 500 or 250, in one or the other nonhorizontal loss ears. Fig. 2 shows how both single frequencies and several different frequencies may be balanced in loudness by this technique.

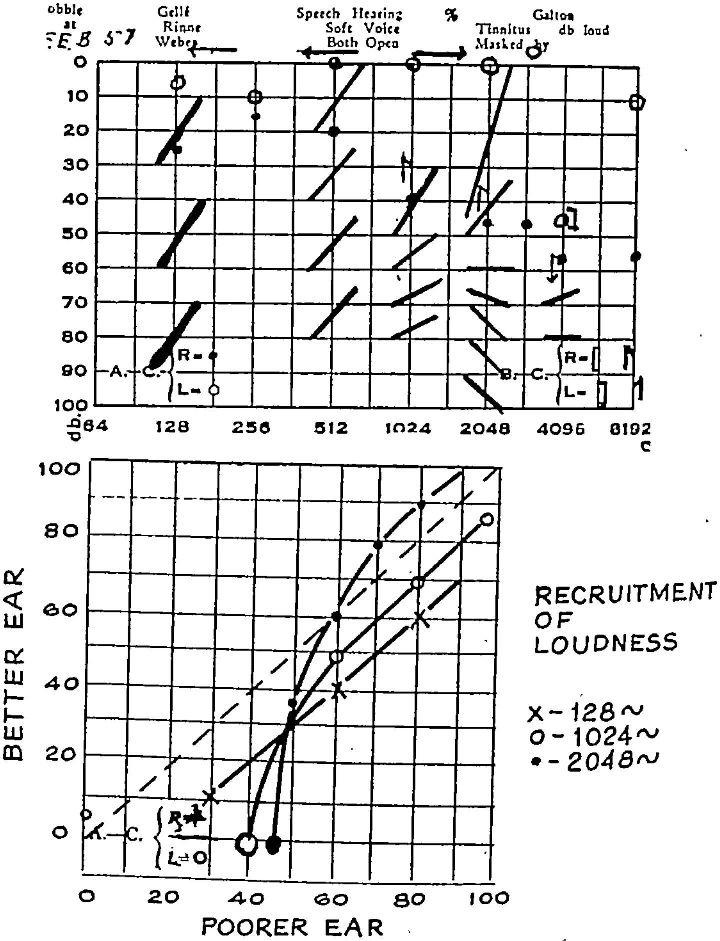


Fig. 3.

Fig. 3 shows the audiogram of a patient with a mixed type of deafness, an impedance deafness in the right ear at the lower frequencies and a neural deafness at the middle and higher frequencies. The recruitment phenomenon is absent in the former and marked in the latter. Notice that there is no recruitment for 128, a slight indication of recruitment for 512, a moderate degree for 1,024, and a marked degree of recruitment for 2,048; also, that at 2,048 there is more than a complete recruitment; there is an over-recruitment. The deaf ear at this frequency senses very loud sounds even louder than the normal ear; also, below the audiogram the equal loudness balances are plotted for three frequencies.

When losses are binaurally equal at the different frequencies, and the audiograms of the two ears are horizontal; only one of the techniques I have just described is available; however, there are several other ways of overcoming the difficulty.

Dr. Georg v. Bekesy[1] has constructed an audiometer to automatically record the recruitment phenomenon. This audiometer presents the tone in constantly increasing steps of 2 db each until the patient indicates he hears it by pressing a button. This pressing of the button causes the intensity to stop rising and immediately to diminish constantly until the patient indicates he no longer hears it by releasing the button. In this way a tracing is obtained which fluctuates from just below to a few decibels over threshold (see Fig. 4). The graphs show wide swings in the tracings (the differential limens for intensity) about threshold in conduction deafness, and very short swings in neural deafness. The extent of the swings also will be influenced by the response time of the patient.

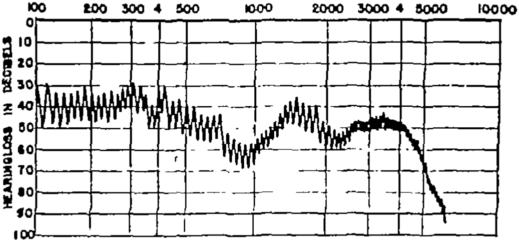


Fig. 4.

Another technique utilizes the fact that the minimum perceptible change (difference limen) in intensity is larger in normal ears or in impeded ears than in ears with neural deafness and, as I have pointed out, becomes progressively smaller with increasing intensity above the pathological threshold. Lüscher and Zwislocki[2] measure the difference limen by using a tone preferably 40 db over threshold and modulating its amplitude, *i.e.*, without interrupting the tone varying the loudness every half second (two fluctuations per second) and gradually lessening the variations in intensity until no change is longer perceptible, until the tone appears continuous and of even timbre. The patient indicates the steadiness or fluctuations in the tone by holding his hand steady or by raising and lowering it as the case may be. The 40 db over threshold is used because loud sounds show less variation with frequency than sounds near threshold, and the amount of recruitment increases even though the ratio declines.

The findings (difference limen) may be expressed either in percentages or decibels and plotted by drawing thick black vertical lines just below the thresholds and superimposed upon the frequency ordinate lines.

Practice enables patients to become more expert and lessens their difference limens.

A neural deafness is presumed when in a case showing more than a 30 db loss the difference limen falls below 8 per cent. Only a very acute normal hearing or impedance deafened observer can detect such a small figure (difference limen).

Another technique is based upon the fact that immediately after the cessation of a sufficiently loud and prolonged tone the threshold of hearing is definitely raised by so-called "auditory fatigue." The threshold shift is greater the closer the testing sound corresponds in frequency with the "fatiguing sound," and the greater the fatigue neural deafness. Huizing[3] showed that immediately following a three-minute stimulation (by 2,000 c.p.s.) at 30 db over the pathological threshold, a threshold shift as much as 12 db may be considered as normal. A threshold shift of over 12 db may be considered as showing the presence of "recruitment" (in neural deafness the shift may be and usually is 19 to 40 db). To be on the safe side and for convenience I use 12½ db.

Since most audiometers have 5 db steps, a 15 db shift may be utilized as the dividing line between normal hearing or impedance deafness and neural deafness. In normal ears or pure impedance deafness there is no increase in loudness values of the testing tone and, therefore, no recruitment, no abnormal fatigue, and no abnormal shift in the threshold. The much greater loudness value obtained in the presence of recruitment explains the greater auditory fatigue produced in neural deafness. Huizing's test technique is simple and gives reliable results. Intensities more than 30 db over pathological threshold should be used with caution because of their large fatigue and possible acoustic trauma effect.

Huizing points out that when, as sometimes occurs, recruitment is absent in the presence of lowered bone conduction, the threshold shift occasioned by masking sounds is within normal limits (not over 12 db). These observations substantiate my own contention that the bone conduction loss under such circumstances should not be interpreted as indicative of a neural deafness; on the contrary, it indicates an inner ear or labyrinth window impedance deafness. In such instances the fenestration operation is not necessarily contraindicated, even though there is a bone conduction loss. It should be clear that it is not the loss in bone conduction which is unreliable in these cases; it is the failure to interpret it properly.

As a rough illustration of a "recruitment" due to loss in capacity, envision two tubes similar in length but differing in capacity (in the ratio of one to three), one 33-1/3 cc., the other 100 cc., the smaller cone shaped; the larger, a cylinder (see Fig. 5).

Quickly fill each tube to 25 per cent of its capacity. Mark this 25 per cent level on vertical lines as indicated in the diagram. Now fill both to 50 per cent of capacity and again mark the levels on the vertical lines. Then fill both to 75 per cent of capacity and mark the levels. Finally fill both to 100 per cent of capacity. Draw straight lines between the similar levels of per cent capacity of each tube. As these lines join the higher and higher equal per cent of capacity levels they

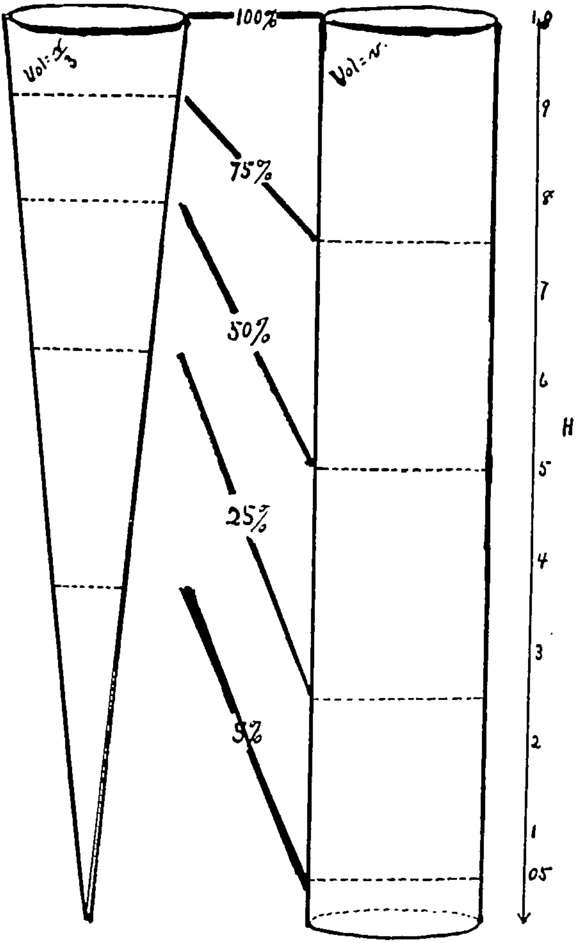


Fig. 5.

make smaller and smaller angles with the horizontal and become shorter and shorter until at 100 per cent the line is the shortest and is horizontal. In other words, with similar percentages of capacity fillings the fluid in the smaller tube, starting with a marked difference in the level, gradually but with decreasing increments approaches and finally reaches the same level as the fluid in the larger tube.

If the neural mechanism senses degrees of loudness in proportion to the per cent of capacity to which it is saturated with sound, and not only in relation to the amount of sound with which we fill it, then the higher the per cent of filling with sound the higher the sensation of fullness. At 100 per cent capacity the sensation of fullness is complete and in the ear this appears to hold true even with marked loss of neural elements, the ultimate number of functioning elements constituting the capacity.

This explanation is helpful but is crude and unsatisfactory from several standpoints, and it does not lend itself to an explanation of over-recruitment of loudness.

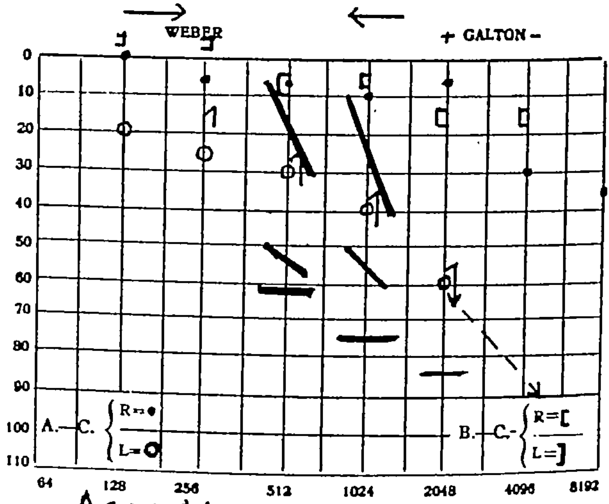
Recruitment is demonstrable in the presence of masking and submasking environmental sounds. Such sounds in a way produce or simulate a neural deafness.

Both "vibratory" and "nonvibratory"[5] tinnitus will evoke recruitment for testing sounds of similar frequency. Not only the illusion of noise called tinnitus, but lesions causing the tinnitus produce neural lesions and, therefore, cause recruitment. Even subaudible tinnitus and subaudible environmental sounds may cause recruitment. Subaudible intensities by summation are superimposed upon similar or other frequencies. In this way a *false threshold* may be obtained. Also, the boosting effects of contralateral sounds suggest the presence of such phenomena. Recruitment is a sensation and is not necessarily exactly the same in different individuals.

As Huizing and others have pointed out, an indication that recruitment is present occurs when patients with neural deafness first try a hearing aid. There is then great difficulty in modulating the voice because of the short distance between the user's transmitter and his mouth. The recruitment not only causes a distortion of speech but also a discrepancy between the loudness of the voice of the hearing aid user and that of speakers some distance away. This may temporarily cause an emotional block so that he is unable to speak.

Recruitment is apparently sometimes absent in old people with neural deafness, but this should not be unexpected because tactile sensations in the aged are well known to be less well differentiated, and hearing is in essence a tactile sensation; moreover, attention may be less concentrated and the technique of the examiner may be at fault. It is often stated that bone conduction is lowered or absent in old people irrespective of the air conduction threshold. This in my experience is not true. One should be careful to inquire as to techniques used and as to the proper interpretation of the bone conduction loss.

Recently, Dix, Hallpike and Hood[1] reported the absence of recruitment in acoustic nerve tumors and explained this on the basis that in such cases there is no cochlear degeneration. They contend that recruitment arises only in lesions of the end-organ and, therefore, its absence in neural deafness provides a test for the differential diagnosis. This is a most interesting observation, and I wish it could be true. I have not as yet observed it, unless there was insufficient hearing to permit the test; however, my material is small. Fig. 6 shows the audiogram of a patient recently tested at my request by Dr. Franz Altmann in the Neurological Institute, Columbia University, before excision of a neurofibroma of the VIIIth nerve. You will note that recruitment is clearly shown.



Acoustic neurinoma

Fig. 6.

Again it should be pointed out that no recruitment occurs in a totally deafened ear, and no recruitment by the binaural loudness technique is apparent if both ears have similar neural lesions; also, that it is at times difficult and even impossible to mask the better ear without contralateral effects, and, moreover, crossed audition may affect the recruitment phenomenon obtained in the ear under test. A neurally deafened ear is hypersensitive to crossed masking because of the recruitment phenomenon which is inherent in it. For these reasons it is usually advisable to avoid masking when testing for "recruitment," and to use it carefully in determining thresholds. It is also pertinent to note that masking sounds may provoke in the contralateral ear a positive Rinne by masking the bone conduction more than the air conduction. It is possible that this may have happened in the case cited by Dix, Hallpike and Hood.

SUMMARY AND CONCLUSIONS.

1. Several techniques of testing for recruitment are described.

2. Recruitment is pathognomonic of neural deafness, but its absence does not necessarily preclude nerve deafness; block lesions may prevent recruitment.

3. Recruitment is greatest immediately above threshold and gradually diminishes in increment as loudness increases. It may appear to be less marked in the lower frequencies than in the higher because neural deafness is usually less marked in the lower frequency areas. As a matter of fact, the lower the frequency the greater the normal increment of loudness.

4. Recruitment may persist even after equal binaural loudness volumes have been reached, so that sounds 110 to 120 db loud may appear to be sensed even louder in the deafened ear than in the normal ear.

5. Recruitment is limited by impedance lesions and, therefore, the two ears, in spite of a recruitment of loudness

because of neural lesions, will always differ in their loudness balance levels by the exact number of decibels which is the measure of their difference in impedance. In this way it is possible to diagnose and measure the amount of the impedance lesions and the neural lesion with a single test.

6. Recruitment for speech is indicated when in moderately deafened ears (overall losses of 20 to 35 db) a 5 or 10 db increase over threshold in the loudness of speech will produce a marked increase in the loudness and intelligibility of the speech (a comfortable level), and more than a 20 db increase will cause distortion and confuse the patient. In obstructive deafness at similar levels a 20 db increase will not do this. Recruitment limits the usable intensity range in hearing aids both because of the speech intelligibility distortion and the fatigue and traumatic factors involved.

7. When recruitment is absent in the presence of markedly diminished bone conduction it indicates that this loss in bone contraction is not due to neural lesions, but is, on the contrary, due to impedance lesions, probably in the oval or round window regions.

8. Recruitment of sensation may be demonstrated in all sensory neural mechanisms.

9. Recruitment tests are valuable for the diagnosis of monaural, very severe or total deafness. (There is no recruitment in a totally deaf ear.)

10. Fatigue, inattention and environmental noise may affect the recruitment tests. Fatigue and noise produce and accentuate recruitment; inattention interferes with the tests for recruitment.

11. Recruitment is valuable as a test for malingering. The malingerer cannot accurately simulate it. If it is present he is not malingering insofar as his neural deafness is concerned.

12. When, as in some instances in otosclerosis bone conduction is lowered the recruitment tests are invaluable to differentiate impedance from neural lesions.

The tests for "recruitment" are reliable and should be employed regularly and with confidence.

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RADIATION THERAPY OF CANCER OF THE LARYNX.*†

B. V. A. LOW-BEER, M.D. (by invitation),
San Francisco, Calif.

The treatment of carcinoma of the larynx is a subject of only the last 75 years of medical history. The first successful attempt to eradicate this lesion by surgical means marks an important milestone in the development of laryngology; however, the initial enthusiasm for successful laryngectomy and laryngofissure was followed by a period in which experience accumulated which led to the realization that surgical eradication of cancer of the throat is a procedure applicable to a limited group of cases. With the advent of radiology around the turn of the century, a new tool was at hand with which to attempt to combat this disease. As early as 1902, attempts were made to treat cancer of the larynx with Roentgen and radium rays. In a survey article, Gradenigo, in 1908, pointed out that radiation does not provide a satisfactory means of treatment, because the technical equipment is inadequate for use in the treatment of cancer of the larynx. It was not until 1922 that the first successful eradication of cancer of the larynx by radiological means was accomplished. Regaud, Coutard and Houtand at this time reported successful treatment of six advanced inoperable carcinomas of the larynx with Roentgen rays. This work not only established radiation therapy of cancer of the larynx but constituted the cornerstone of radiation therapy as a medical specialty. It was Regaud's experimental work carried out in 1914 which was the basis of the method employed in the treatment of these six patients. Inserting radium needles into rams' testes, he demonstrated

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif, May 25, 1950.

†From the Division of Radiology, University of California School of Medicine, San Francisco, Calif.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

that doses of low intensity applied over prolonged periods resulted in a more complete destruction of cells in active mitosis than is possible with high intensity, short term irradiation. He also found that low intensity irradiation carried out over a prolonged period of time results in a better protection of the adjacent tissues than is possible when high intensity short term irradiation is employed. This principle, which was first applied to the use of X-rays by Coutard and which is generally and correctly known as the "Coutard method," is based on treating the cancerous lesion over a period of several weeks, by exposing the lesion to Roentgen rays daily to fractions of the total dose over several hours. He contended that by fractionation and protraction of the radiation dose, it is possible to deliver a much higher total quantity of radiation than is possible with single dose, high intensity irradiation. Coutard's original method of treating carcinoma of the larynx consisted of giving 100 to 150 r. in a period of two to four hours daily over three to four weeks' time. He used 140 to 160 kv. generated X-rays and filtration varying from 10 mm. to 15 mm. aluminum. By this means he was able to provide highly penetrating radiation which in Coutard's experience produced less effect on the skin than any radiation quality theretofore used in radiological treatment. In considering Coutard's original method in retrospect, it is obvious that the great merit of Coutard's achievement lies in his recognition of the significance of fractionation of the radiation dose and heavy filtration of the X-ray beam. Protraction was an unavoidable necessity with the technical equipment at Coutard's disposal, and it has subsequently been shown to be of secondary importance; however, be that as it may, despite various modifications of the technique originally suggested by Coutard, the fundamental idea of delivering a carcinocidal dose of 5,000 to 7,000 r. in a single series of daily fractions over a limited period of three to six weeks still controls radiological practice in the treatment of laryngeal cancer.

It is my privilege to present to you our experience in treating cancer of the larynx in the division of radiology at the University of California Hospital from 1931 to the present time. Until the end of 1942 this work was carried on by

Dr. Robert S. Stone, who as you know was called to head the health protection program of the Manhattan Project shortly after the outbreak of the war. The responsibility of carrying on radiation therapy has devolved upon me since 1943. During the whole period under consideration the work has gained immeasurably from the close cooperation between the divisions of otolaryngology and radiology; especially from the constructive and helpful attitude of the chairman of the division of otorhinolaryngology, Dr. Lewis Morrison, and the great interest of your president, Dr. Robert C. Martin. Dr. Morrison has been interested not only in the clinical aspects of the problem but also in the physical and biological problems of radiation. His understanding of radiological problems has contributed much to the development of our ideas in the radiological treatment of laryngeal cancer.

SELECTION OF CASES.

In the nearly 20-year period since 1931, a total of 224 patients with proven laryngeal cancer were seen in the division of radiology. Five of these were examined but for various reasons were not treated in our department. Sixteen patients received radiation therapy for surgical recurrences. Twenty patients, in almost preterminal stages of their disease, received not more than two or three treatments and, therefore, cannot be included as part of a group in which the therapeutic value of radiation procedure is to be analyzed. The follow-up is incomplete for 10 patients. Nine of these were treated between 1931 and 1940. Sixteen patients were treated with neutrons. Patients who received X-ray treatment postoperatively, or for surgical recurrences, or in whom surgery could not be carried out to the extent originally intended, we have excluded from our discussion because we are concerned here with evaluation of X-ray therapy as the primary method of treatment in cancer of the larynx. One hundred fifty-seven patients received a full course of X-ray treatment as a primary therapeutic procedure, complete follow-up has been obtained on all of them, and these patients are the subject of our analysis today. Of the 157 completed cases, 140 were males and 17 were females. Age distribution of the patients is shown in Table 1. The youngest patient was 35 years old, the oldest 81 years old at time of treatment.

TABLE 1.

CARCINOMA OF THE LARYNX

INCIDENCE BY AGE AND SEX OF 157 X-RAY TREATED CASES

AGE	SEX		TOTAL
	MALE	FEMALE	
Under 40	3	–	3
41–50	25	7	32
51–60	50	3	53
61–70	48	5	53
71–80	12	2	14
Over 80	2	–	2
TOTAL	140	17	157

No restrictions were established for acceptance of patients for radiation therapy beyond the microscopical diagnosis of carcinoma. The patients were classified by the primary site of origin of the lesion regardless of subsequent extension, that is, the anatomical classification rather than the surgical classification or definition of intrinsic and extrinsic lesion was followed. We were fully aware of the confusion which results from such classification and from the fact that one is easily persuaded to interchange the anatomical and surgical meaning of the definition of intrinsic and extrinsic laryngeal lesion. In the analysis which I am presenting to you, tumors are not graded according to cellular differentiation. Of the 157 lesions, 153 were squamous cell carcinoma and four were adenocarcinoma. While little attention was paid to cellular differentiation, great attention was paid to the location and extent of the lesion, evidence of inflammation and infection, and the presence of metastases. Of the 18 lesions treated in the period between 1931 and 1935, 10 were intrinsic and eight extrinsic. Of the 24 lesions treated between 1936 and 1940, 16 were intrinsic and eight were extrinsic. Of the 59 lesions treated between 1941 and 1945, 47 were intrinsic and 12 extrinsic. Of the 56 lesions treated between 1946 and the present time, 44 were intrinsic and 12 were extrinsic.

TABLE 2.

CARCINOMA OF THE LARYNX

SURVIVAL BY CLASSIFICATION INTO INTRINSIC AND EXTRINSIC LESION

SURVIVAL YEARS	1931 – 1935		1936 – 1940		1941 – 1945		1946 – 1950	
	INTRINSIC	EXTRINSIC	INTRINSIC	EXTRINSIC	INTRINSIC	EXTRINSIC	INTRINSIC	EXTRINSIC
0 – 1	6	5	8	4*	11°°	9**	11●	6
1 – 2	1	2*	1	3	5°°	1	9●●	3
2 – 3	1	1	–	–	1	1	10●●●	1
3 – 4	1	–	1	1*	2*	–	4●●●●	2
4 – 5	–	–	1	–	h7°	–	10●●●●●	–
5	1	–	5	–	hh21°	1	–	–
TOTAL	10	8	16	8	47	12	44	12

* One died of intercurrent disease
** Two died of distant metastases
° Alive and well
°° One early
● Six early

●● Four early
●●● Seven early
●●●● Two early
●●●●● Seven early
h All early
hh Eight early

PERIODS OF OBSERVATION.

We have divided our observation into four time periods: the first, from 1931 to 1935, inclusive, the second from 1936 to 1940, the third from 1941 to 1945, and the fourth from 1946 to the present time. We have felt justified in dividing our observation period of 19 years for the following reasons: Dr. Robert Stone, chairman of the division of radiology, joined the University of California hospital staff in 1928. At that time no deep X-ray therapy was available in the department. The first 200 kv. therapeutic X-ray machine was installed in 1931. Two years later, through the cooperation of Dr. Stone, Dr. Ernest Lawrence and Dr. Sloan, a one million volt unit was built and put into operation in 1933. Accordingly, we think of the period from 1931 to 1935 as the pioneering period in Roentgen therapy in our department. A total of 18 patients, most of them with advanced laryngeal cancer, were treated in this period in an attempt to eliminate the malignant lesion locally by means of radiation.

The second period, that is, from 1936 to 1940, I should call the period of experimentation. The physical and biological effects of supervoltage radiation were studied. Comparison of effects of high voltage (200 kv. generated X-rays) and super voltage (one million generated X-rays) upon the skin and the underlying tissues held the center of interest. This period also includes experimentation concerning daily doses, total doses and time factor of fractionation. In this period a total of 24 learyngeal cancers were treated. Although the majority were still advanced lesions, some early lesions, namely, those localized to the true vocal cord, as ascertained by indirect or direct laryngoscopy, were also referred for primary radiation therapy.

The third five-year period including the years 1941 to 1945 is characterized by a systematic approach to the problem. In this period a definite pattern of procedure was developed relative to the localization of the lesion, extent of the lesion and site or origin of the lesion; although strictest individualization was carried out in every case. In the period between 1941 and 1945, a total of 59 patients were treated.

Let me call the last period, since 1946, the period of modern expansion of the whole field of radiology. In this last period between January, 1946, and December, 1949, a total of 56 patients received primary radiation therapy for laryngeal cancer.

TABLE 3.

PATIENTS TREATED 1941-1945

SURVIVAL	1000 Kv	200 Kv	COMBINATION		TOTAL
			A	B	
0-1	15	3	2		20
1-2	2		3	1	6
2-3			1	1	2
3-4	2				2
4-5	2		2	3	7
Over 5	6		13	3	22
					59

TABLE 4.

PATIENTS TREATED 1946-1949

SURVIVAL	1000 Kv	200 Kv	COMBINATION		TOTAL
			A	B	
0-1	4	12	1		17
1-2		8	2	2	12
2-3		10	1		11
3-4	2		2	2	6
4-5	7		3		10
					56

FACTORS OF TREATMENT.

The 157 patients who comprise the entire group of cases who received complete treatment with X-ray as the primary therapy were treated either with high voltage or with super voltage radiation. It was always the intention to carry through the complete treatment with only one quality of radiation; however, in a number of instances, because of mechanical breakdown of one or the other equipment, it became necessary to treat the patients with both machines and thus with two different qualities of radiation — high voltage and super voltage radiation. The high voltage radiation consisted of X-rays generated by 200 kv. potential. Quality of the radiation in the 19 years has varied from 0.95 mm. copper to 1.3 mm. copper half-value layer. Focal skin distance was either 50 cb. or 80 cm. and the minute output varied from 45 r. to 50 r. for 50 cm. and from 15 r. to 19 r. for 80 cm. measured in air. Super voltage radiation consisted of X-rays generated by one million volt potential. The beam was filtered with 2.2 mm. of lead, 0.6 mm. tin, 1 mm. copper and 1.5 mm. aluminum. The quality of the radiation was that of 9 mm. copper half-value layer. The machine was operated at 2.5 or 3 milliampere intensities. The minute output varied between 19 and 22 r. measured in air at 70 cm. Treatment ports varied considerably from 5 x 5 to 10 x 15 cm. Fields were always square. Patients were treated in sitting position with the head fixed on a head rest and held in place by a head band. Positioning was always done by a senior radiologist. Field films were taken of the therapy setup to check position. On the super voltage machine positioning was checked with the aid of an exit beam localizer, on the high voltage machine, by visual positioning. Treatments were given daily except on Sundays. The daily dose fractions varied. Patients who were treated with high voltage X-rays received a maximum daily dose of 300 r. measured in air which corresponds to a tissue dose at 5 cm. depth for a 5 x 5 cm. field approximately 160 r. Patients treated with super voltage X-rays received daily doses up to 400 r. measured in air corresponding to a tissue dose at 5 cm. depth for a 5 x 5 cm. field to approximately 220 r. One hundred forty-one patients received treatment through two opposing lateral neck fields, 12 patients were treated through a single anterior field, and in four patients who were treated in the first five-year period, three treatment ports were used, namely, two lateral ports and one posterior neck port. The treatment course varied between 18 to 25 days in the first and second periods but was extended up to 36 to 38 days in the third and fourth periods of our observations. Most of the patients since 1941 were treated in a period between 26 and 32 days.

The calculated tumor dose delivered in the first five-year period did not exceed 4,000 r. In the second five-year period the treatment dose varied between 4,000 r. and 6,000 r.; most of the patients received around 5,000 r. One patient in this period received 8,000 r. tumor dose and he is the patient with the longest survival of the whole group. In the period from 1941 to 1945 the tissue dose varied between 5,500 and 7,500 r. Patients were treated in a period between 28 and 35 days. Two patients received a tissue dose of 7,800 and 8,100 r., respectively, with super voltage radiation.

From 1946 to the present the tissue dose varied between 5,500 r. and 7,000 r.; the majority of patients received around 6,000 r. tissue dose with high voltage and around 6,500 r. with super voltage radiation. The treatment period extends over 32 to 38 days.

TABLE 5.

CARCINOMA OF THE LARYNX
SURVIVAL OF PATIENTS BY YEAR AND PERSISTENCE OF LESION

YEAR	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49
NO. PATIENTS TREATED	3	4	2	6	3	6	4	4	4	6	2	8	12	17	20	19	12	16	9
LESS THAN 1 YR. WITHOUT CA.					1					1			1		1		1		4
WITH CANCER	2	3	2	2	1	2	3	2	1	3	1	4	1	9	3	2	6	3	1
1-2 YEARS WITHOUT CA.							1						1				1	6	4
WITH CANCER	1	1		1		1			1	1		1	2	1	1			1	
2-3 YEARS WITHOUT CA.																1	4	6	
WITH CANCER				1	1							2							
3-4 YEARS WITHOUT CA.				1		1										6			
WITH CANCER						1					1			1					
4-5 YEARS WITHOUT CA.														1	6	10			
WITH CANCER										1									
OVER 5 YEARS WITHOUT CA.				1		1		2	2			1	7	5	9				
WITH CANCER																			
TOTAL	18					24					59					56			

Analysis of the physical factors of X-ray irradiation reveals that in the period 1931 to 1935, five patients were treated with super voltage radiation, 10 were treated with high voltage radiation, and three were treated with a combination of super and high voltage radiation. One patient was treated predominantly with high voltage, two were treated predominantly with super voltage. Table 6 shows survival for this group.

TABLE 6.

PATIENTS TREATED 1931-1935

SURVIVAL	1000 Kv	200 Kv.	COMBINATION A*	COMBINATION B**	TOTAL
Under 1 Yr.	2	6	2	1	11
1-2		3			3
2-3	2				2
3-4	1				1
4-5					
Over 5		1			1
					18

* Predominantly supervoltage
** Predominantly high voltage

Twenty-four patients received X-ray treatment between 1936 and 1940. Sixteen received super voltage radiation, three received high voltage radiation, and five received a combination of both. In four cases radiation was predominantly super voltage, in the remaining case it was predominantly high voltage. Table 7 shows survival for the group.

TABLE 7.

PATIENTS TREATED 1936-1940

SURVIVAL	1000 Kv	200 Kv	COMBINATION A	COMBINATION B	TOTAL
Under 1 Yr.	8	1	2	1	12
1-2	3		1		4
2-3					
3-4	1	1			2
4-5	1				1
Over 5	3	1	1		5
					24

Fifty-nine patients received X-ray treatment between 1941 and 1945. Twenty-seven received super voltage radiation, three received high voltage radiation and 29 received a combination of both. Twenty-one received predominantly super voltage and eight received predominantly high voltage. Table 8 shows survival of the group.

Fifty-six patients received X-ray treatment from 1946 to the present. Thirteen received super voltage radiation and 30 received high voltage radiation. Thirteen received a combination of both, with super voltage predominating in nine cases and high voltage predominating in four cases.

NEUTRON THERAPY.

The 16 patients treated with neutrons by Dr. Stone have been observed by me since 1943. All of them received neutrons generated in the 60-inch cyclotron by interaction of 16 Mev. deuterons with a beryllium target. Treatment was given by the fractionation method, and wherever possible by crossfire techniques. The rate of administration was around 5 n. per minute measured on the 100 r. chamber of the Victoreen condenser r. meter. The cyclotron operated erratically, making it necessary to vary the rate of administration, the daily doses, the number of fields treated per day, the total dose, and the number of days between the first and last treatment. Of the 16 patients, only two had lesions which could be called early lesions. One of these patients has survived for nine years and is well and active with a well preserved voice. In all the other patients the lesions were very extensive and definite metastases were present; many of the lesions were so extensive that it was impossible to distinguish between extension and regional metastases. Four of these patients received an *incomplete* course of neutron therapy. Of those who received a complete course, nine lived less than one year; two lived between one and two years and one, as I mentioned, is still living. This patient received 18 treatments in 48 days. The total dose was 509 n. I mention these neutron treated patients in order to show you the skin reactions immediately after treatment and eight years later in the patient who survived. It is obvious that the reaction was considerably more intensive immediately following treatment than that observed with high voltage or super voltage X-rays. The skin and subcutaneous tissue changes manifested as a late reaction two years after radiation with neutrons was considerably more intensive than we have seen with the highest dose ever given by us with super voltage or high voltage radiation. It may be mentioned that in the seven autopsy examinations in patients who had received neutron therapy, no tumor cells were found in the treated areas, but in all of these patients there were deep necrotic ulcers.

REACTION TO RADIATION.

The radiation reaction on the skin varied from moderate scaling to marked bullous epidermolysis and on the mucous membranes of the larynx from moderate mucositis to pseudomembranous mucosal reaction. The highest single field dose was 9,200 r. delivered in a period of 33 days. Color slides show early and late skin reactions following X-ray therapy in patients treated through a single port or through two "crossfire" ports in the period between 1941 and 1946. First marked reaction usually appeared around the twelfth to fourteenth day of treatment with high voltage radiation, while with super voltage radiation first marked reactions were delayed to the sixteenth to eighteenth day, when the whole treatment course was delivered in 28 to 32 days. Appearance of the reaction was generally in direct relation to the daily dose fractions. It was observed that the same skin effect was produced by approximately 20 to 25 per cent higher individual and total doses with super voltage radiation than with high voltage radiation. Mucosal changes were also less from super voltage radiation than from the same quantity of high voltage radiation. The reaction reached its height approximately five to seven days after conclusion of treatment and subsided usually in four to eight weeks after conclusion of treatment. The critical reaction period usually extended over a period of approximately 10 days. Five per cent boric acid ointment and/or 1 per cent benzocaine ointment was used to combat the acute reaction of the skin. Reaction of deep-seated tissue such as cartilage may result immediately following or several years after radiation therapy. Perichondritis occurs in almost all advanced carcinoma of the larynx and may result early or late following radiation therapy. Approximately 2.5 per cent of our cases have developed perichondritis to some degree. In most instances it has coincided with persistence of tumor. Perichondritis is rarely caused by radiation alone, but is due to infection and inflammation of the tumor bed where the tumor infiltrates the vascular connective tissue, the lymphatics or invades the cartilage. In addition to starting the treatment with small initial doses of radiation, antibiotics should be employed in patients treated with radiation therapy during treatment and for approximately six weeks after radiation therapy. Occasionally almost complete regeneration of cartilage destroyed by tumor and inflammation may follow successful radiation therapy.

SURVIVAL ANALYSIS.

Of the 10 intrinsic lesions treated between 1931 and 1935, one patient has survived more than five years and is free of cancer. This patient was treated with high voltage radiation. One of the patients with extrinsic lesion treated in this period survived longer than two years.

Of the 16 patients with intrinsic lesions treated from 1936 to 1940, five, or 31.2 per cent, have survived more than five years free of cancer. Six patients, or 37.4 per cent, have survived more than four years free of cancer. Seven patients, or 43.7 per cent, have survived more than three years. Of the eight extrinsic lesions treated in this period, seven patients died within two years and one died in the fourth year after treatment.

Of the 47 intrinsic lesions treated from 1941 to 1945, 21 patients, or 45.8 per cent, have survived more than five years free of cancer. Twenty-eight patients, or 59.5 per cent, survived more than four years, 30 patients, or 64 per cent, have survived more than three years. Nineteen patients had early lesions localized to one cord. Of these, 15, or 79.2 per cent, have survived more than four years.

Of the 12 extrinsic lesions treated in this period one patient with primary cancer of the epiglottis had survived more than five years free of cancer.

TABLE 8.

SURVIVAL DATA AND CAUSE OF DEATH

Time of Survival	Patients Alive	Patients with Ca or Regional Metastases	Patients Dead of Distant Metastases	Patients Dead of Intercurrent Disease	TOTAL
0-1 Yr.	5	50		5	60
1-2 Yrs	10	12		3	25
2-3 Yrs	11	4			15
3-4 Yrs	6	2	1	2	11
4-5 Yrs	16	1		1	18
Over 5 Yrs	26	0	1	1	28

Fifty-six patients were treated for laryngeal cancer from 1946 to the present time. Ten of these patients are alive more than four years after treatment and are free of cancer. Six patients are living more than three years after treatment and are free of cancer. Eleven patients are living more than two years after treatment and are free of cancer. Twelve patients are living more than one year after treatment. One of these patients has local cancer, the others are free of cancer. Twelve patients survived treatment less than one year. Of these 56 patients, 44 had intrinsic lesions.

DISCUSSION.

In the past 20 years since Coutard's presentation of his paper at the Otorhinolaryngological Convention in 1931, reports of more than 2,000 patients primarily treated with radiation are available. The statistical figures given by authors from different centers vary widely. When one tries to explain these wide variations of statistical data presented by expert investigators it becomes obvious that one reason for these is the lack of a common basis for evaluation of the cases, due to nonuniform classification of the lesions treated. As has been stated before, classification into intrinsic and extrinsic laryngeal tumors is entirely unsatisfactory, and gives rise to confusion and ambiguity. Comparison of neoplasms arising in various parts of the anatomical larynx reveals entirely different biological entities. Tumors of the true cord, for instance, grow slowly, metastasize rarely, while those arising on the false cord or ventricles grow more rapidly and metastasize early. Tumors of the aryepiglottic fold, the pyriform fossa are not laryngeal tumors at all, either biologically or histogenetically.

Plans to introduce a classification based on the site of origin of a malignant lesion were attempted by Frenckner, in 1935, and by Lederman, in 1943. For the last year and a half, I have tried to formulate my own ideas concerning classification by site of the tumor which would satisfy the radiation therapist as well as the laryngologist and facilitate a better comparative analysis of treatment procedures among various centers here and abroad. I am taking this opportunity to present to you a plan of classification for your consideration in the hope that it may be adopted by your Society, and that such a step may lead to the establishment of an international classification of laryngeal cancers similar to that so generally adopted for cancers arising from the uterine cervix.

I have divided tumors of the larynx into two main groups: Group I includes tumors which arise from the true cord, and Group II includes tumors which arise on the ventricle, false cord, aryepiglottic fold, the epiglottis, subglottic area and pyriform fossa.

Tumors which arise on the true cord are divided into four stages:

Stage I: Involvement of not more than two-thirds of one vocal cord without impairment of mobility.

Stage II: Involvement of one cord and anterior commissure or part of both cords, without impairment of mobility.

Stages I and II are further subdivided into Group A: proliferative exophitic, and Group B: infiltrative-ulcerative.

Stage III: Tumors extend beyond the vocal cord involving the ventricle, false cord, arytenoid and/or subglottic region. Mobility is impaired partially or completely.

Stage IV: Tumor involves the true and false cords, arytenoids, and/or epiglottis. Mobility is impaired completely. Cervical metastases are usually present.

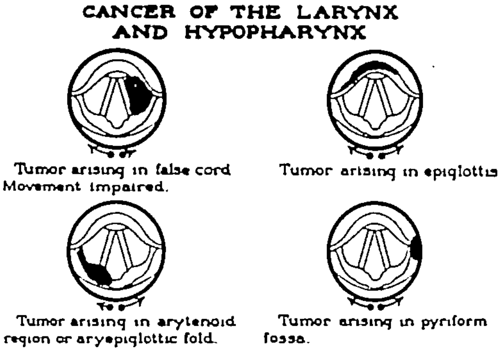
CANCER OF THE LARYNX AND HYPOPHARYNX
Tumor arising in false cord Movement impaired.
Tumor arising in epiglottis
Tumor arising in arytenoid region or aryepiglottic fold.
Tumor arising in pyriform fossa.

Fig. 1.

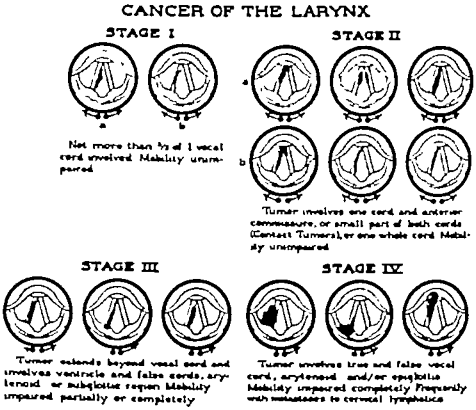
CANCER OF THE LARYNX
STAGE I
a
b
Not more than ½ of 1 vocal cord involved Mobility unimpaired
STAGE II
a
b
Tumor involves one cord and anterior commissure, or small part of both cords (Contact Tumors), or one whole cord Mobility unimpaired
STAGE III
Tumor extends beyond vocal cord and involves ventricle and false cords, arytenoid or subglottic region Mobility impaired partially or completely
STAGE IV
Tumor involves true and false vocal cord, arytenoid and/or epiglottis Mobility impaired completely Frequently with metastases to cervical lymphatics

Fig. 2.

In our analysis we have paid little attention to the cellular differentiation or cell grading of the tumor, despite the fact that many authors consider cellular differentiation important in determining indication for radiation therapy, in deciding on management of treatment with respect to daily dose fractions and total dose, and in predicting response to radiation therapy. In my experience cellular differentiation is not an adequate basis for such decisions. It is often contended that lack of cellular differentiation is synonymous with radiosensitivity, that the tumor mass may disappear rapidly as all the irradiated cells respond almost at once. While this may be correct, it is a mistake to confuse radiosensitivity with radiocurability. Radiocurability is measured by absence of recurrence in five-year period. Admittedly this definition is subject to discussion. In general, it may be stated that patients with laryngeal cancer who survive four to five years without recurrence very rarely succumb to the local disease at a later time. They may develop second malignancies, particularly of the mucosal linings at other sites, but very rarely do they develop metastases from the primary laryngeal carcinoma. The five-year "cure" rate in general is in good correlation with the average growth coefficient, that is, the rate of growth of laryngeal tumors. The growth coefficient is also closely related to the tendency to regional metastases. Lymph node involvement reduces radiocurability because tumors with a tendency to early lymph node involvement differ in their biology from those which tend to metastasize late. Undifferentiated anaplastic tumors have a high growth coefficient and tendency to spread. From the point of view of five-year survival they are most frequently incurable. It is, therefore, obvious that the two terms, radiosensitivity and radiocurability are not interchangeable. On the contrary, they represent diametrically opposite biological behavior of the tumor relative to a therapeutic agent. It is also true that the degree of cellular differentiation may not be uniform throughout the tumor or in the primary tumor and in the involved lymph nodes. A biopsy taken from one area of the tumor or from a lymph node, therefore, may fail to represent accurately the chief cellular differentiation of the tumor as a whole. For these reasons I am unable to agree with some of my colleagues who pay so much attention to cellular appearance of the biopsy specimen in conducting treatment, deciding on treatment method and predicting the outcome of therapy. On the other hand, I believe that clinical classification of the tumor into proliferative-exophitic and infiltrative-ulcerative is a valid guide in carrying out radiation therapy. Exophitic tumors, even when large, are prognostically more favorable than infiltrative-ulcerative tumors even when the latter are of small size. Ulcerative-infiltrative tumors interact with the tumor bed much more than exophitic-proliferative tumors and so affect primarily the vascular connective tissue of the tumor tion must be taken into consideration in every individual instance. Personally I believe that delivering an assumed carcinocidal dose of 5,500 r. to 7,000 r. in a period of less than 28 days produces a too severe tissue reaction; similarly, extension of treatment beyond 35 to 38 days is not desirable because of probability of cell recovery on the one hand and connective tissue damage on the other. Some inflammation is observed in most laryngeal cancers. For this reason, it is our practice to institute radiation therapy with doses which do not exceed 50 to 75 r. calculated on the larynx during the first two to four days. In this way it usually is possible to avoid initial edema of the larynx which may necessitate tracheotomy. Although tracheotomy does not contraindicate radiation therapy, it does interfere with the radiological management of the patient.

Coutard in his original work advocated low intensity, which is synonymous with protraction of treatment. Neither experimental work nor clinical experience has confirmed the validity of this theory. In our practice we treat one field a day in periods lasting from 10 to 25 minutes. In my own view there may be merit in treating the patient twice daily with smaller individual doses. It is recognized, however, that such a procedure would offer practical difficulties for most patients and to most institutions.

Of equal importance with the physical factors is the exact positioning of the patient. Neglect or errors in positioning may result in overdosage to some areas and underdosage to others, either of which may prove to be the crucial factor in success or failure of the treatment. The importance of this consideration is due in part to many treatment days involved in a full course of therapy. It is necessary to keep the patient under constant surveillance during treatment to be sure that his position does not change during exposure.

It is usual in most centers to treat laryngeal cancers through two opposing crossfiring fields. From the point of view of delivery of a uniform dose this technique leaves something to be desired. Unavoidably, areas of overirradiation, so-called "hot spots," occur, as has been shown by Mayneord and other British investigators. To some extent this can be avoided by homogenizing the tissue volume under irradiation, which can be accomplished with special treatment cones, rice bags or other devices. We have treated 12 patients experimentally through a single anterior field, and the patient who received the highest dose by this method, namely, 8,200 r. calculated on the larynx, is alive and well, more than five years after treatment with very little skin changes and practically no subcutaneous sclerosis. Another patient who received 5,500 r. tissue dose also by this method developed a perichondritis with all the usual complications. It is our conclusion that when single field treatment is employed the field size should not exceed 3 cm. in diameter when a round field is used or 3 x 3 cm. for a square field. Accordingly, we believe that single-field treatment should be limited to very small, localized tumors. At present we are interested in multiple field and rotational therapy. By placing the patient in such a position that the larynx is in the rotational axis, a very narrow treatment beam can be employed, which facilitates delivery of a dose to the tumor with such great accuracy that a differential of more than five to one between the tumor and the tissue may be attained.

In giving the necessary close attention to the physical aspects of the treatment, one must not, of course, overlook the needs of the whole organism of which the tumor is only a small part. Radiation therapy is an exhausting procedure for the patient. Consequently, preliminary examination to determine the patient's general condition is of great importance. This must include examination of the cardiovascular system, lungs and kidneys. A Wassermann reaction should be taken since tissue healing is markedly impaired in active luetic patients. In case of active lues, antiluetic treatment should be carried on simultaneously with radiation therapy. During treatment, daily examination is of great importance. The patient's nutrition must be carefully supervised. It is preferable to hospitalize the patient in the fourth week of treatment and to keep him in the hospital for one or two weeks after the conclusion of treatment. Weekly follow-up examinations for the first six weeks after conclusion of treatment, and then biweekly examination for the next three months should be the rule. If untoward effects develop in this period and are recognized early, surgical intervention may be carried out easily. In my opinion, it is ill advised to repeat a course of radiation therapy after failure.

It is my strong belief, based upon the experience which I have described, that when all precautions and safeguards, both physical and clinical, are observed, radiation therapy is a satisfactory means of treating laryngeal cancer. In early cases limited to the vocal cord, I believe that radiation is the treatment of choice. Radiation properly administered leaves the larynx with the least anatomical or functional impairment. Restricting radiation therapy to advanced cases or to treatment of postoperative recurrences or the palliation of hopeless cases is not warranted in the light of modern results. Admittedly, there is room for further improvement of radiological techniques and in view of development in the field of radiation, we may anticipate further great improvement in the use of X-rays in the not far distant future.

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BOOK REVIEW.

Hearing Tests and Hearing Instruments. Leland A. Watson and Thomas Tolan, M.D., Baltimore, Md.: The Williams & Wilkins Co., 1949, 597 pp. $7.00.

This book contains the following six major parts: Backgrounds of the Audiometer, Interpretations of Audiometric Data, Technical and Engineering Aspects of the Audiometer, Social Aspects of Audiometry, Hearing Aids and Advanced Audiometry. It includes an elaborate bibliography and a glossary of terms in audiology. The stated object of the book is to provide a comprehensive and primarily practical text on hearing instruments and their application. It is intended to serve physicians, school nurses, rehabilitation officers and those who dispense and fit hearing aids.

The authors present a rich body of information from a wide variety of sources hitherto unavailable in compact form. Certainly the extensive bibliography, although unorthodoxly arranged, constitutes an extremely helpful guide for those probing deeply into the problems of hearing and deafness.

This reviewer questions whether the book can be regarded as a definitive "text" in the field. The levels of difficulty of the material undulate disconcertingly from relatively elementary concepts to concepts which would elude even sophisticated clinicians. The authors imply that this happens, but they should be aware that a "text" also implies a reasonable degree of uniformity.

The equivalence assigned to widely differing (in amount, kind and accuracy) information to support certain points of view is also disturbing when it appears in a "text." Carefully gathered laboratory data are implicitly refuted by subjective opinions, the validation for which is not apparent.

The repeated appearance on audiogram charts and batteries of the name of the firm with which one of the authoris connected serves no useful purpose and certainly detracts from the objectivity or disinterestedness for which a "text" in a scientific field should strive.

The book contains much that is helpful but the reader should evaluate the material critically. This would be a useful exercise for anyone interested in hearing, including the "experts."

S. R.S.

MPORTANT NEW PRODUCT ANNOUNCED FOR THE TREATMENT OF SINUSITIS.

Another new member of the furacin(R) family of topical ntibacterial preparations is announced by Eaton Laborato-es, Inc.: Furacin Nasal with Ephedrine. This special aque-us solution of furacin with ephedrine is indicated for the eatment of sinusitis and rhinitis of bacterial origin.

Furacin Nasal was evaluated on over 300 patients at a ading medical center. Many cases of chronic sinusitis and hinitis were cured; cases of chronic atrophic rhinitis were arkedly benefited. Many gram-negative and gram-positive acteria proved susceptible to this preparation. Sensitization ccurred in only one of these 300 patients.

Other clinical preparations of furacin, brand of nitrofura-one N. N. R., now include Furacin Soluble Dressing, Furacin olution, Furacin Anhydrous Ear Solution and Furacin Vagi-al Suppositories.

Furacin Nasal with Ephedrine is supplied in dropper bot-es of 30 cc. in cartons with slip labels.

MISSISSIPPI VALLEY MEDICAL SOCIETY MEETING AT SPRINGFIELD, ILL., SEPT. 27, 28, 29, 1950.

The fifteenth annual meeting, Mississippi Valley Medical ociety, will be held at the Elks Club, Springfield, Ill., Sept. 7, 28, 29 under the presidency of Dr. N. G. Alcock, of Iowa ity, Iowa, immediate past-president of the Iowa State Medi-l Society. Over 30 clinical teachers from the leading medical hools will conduct this great postgraduate assembly whose ntire program is planned to appeal to general practitioners. here will be over 50 scientific and technical exhibits, noon ound table lunchons, etc. *No registration fee* will be charged nd every ethical physician is cordially invited and urged to ttend. The entire program and all exhibits will be held in e newly remodeled, air-conditioned Elks Club of Springfield. he American Medical Writers' Association will hold its nnual meeting at the Elks Club on Sept. 27. Programs of oth meetings may be obtained from Dr. Harold Swanberg, ecretary, M. V. M. Society and A. M. W. Association, 209-24 W. C. U. Building, Quincy, Ill.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 108BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc.. 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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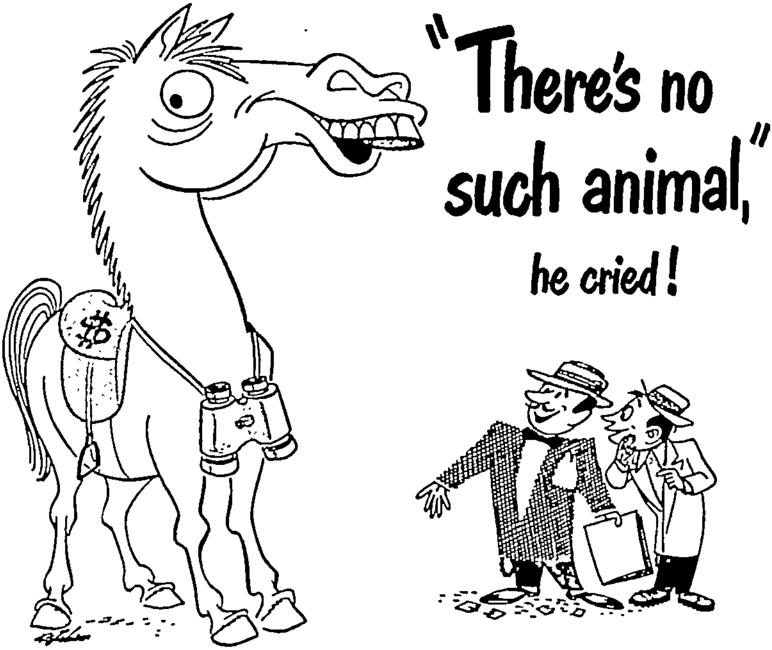
Pro-Tesorero: Dr. Jorge Couzo.

"There's no such animal," he cried!



MY FRIEND and I were picking the ponies one day when I started telling him about *a sure thing* I heard about.

"You say it pays four bucks for every three?" he asked. "And can't lose? It *automatically* wins? Must be illegal!"

"Not a bit," I replied. "In fact, the government very much approves..."

"Our government approves of a horse who can't lose..."

"Who said anything about a horse?" I asked.

"So what else could it be but a horse...?"

"It not only could be—but is—U. S. Savings Bonds," was my prompt reply.

"For every three dollars you invest in U. S. Savings Bonds you get four dollars back after only ten years. And if you're a member of the Payroll Savings Plan—which means you buy bonds *automatically* from your paycheck — that can amount to an awful lot of money while you're not looking. Hey, what are you doing?"

"Tearing up my racing form! The horse I'm betting on from now on is U. S. Savings Bonds."

Automatic saving is sure saving—U.S. Savings Bonds

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THE
LARYNGOSCOPE.

VOL. LX AUGUST, 1950. No. 8

NEW INVESTIGATIONS ON THE PHYSIOLOGY OF THE LABYRINTHINE FLUIDS.*†

FRANZ ALTMANN, M.D., and JULES G. WALTNER, M.D.,
New York, N. Y.

Three years ago we reported before this Society the results of preliminary experiments in rabbits designed to investigate some questions pertaining to the physiology of the labyrinthine fluids. Using the method employed by Weed in his studies of the resorption of the cerebrospinal fluid, small amounts of solutions of iron-ammonium citrate and potassium ferrocyanide were introduced into the subarachnoid space. The animals were killed 15 to 120 minutes after the injection and the temporal bones fixed in formalin-hydrochloric acid solutions. Since the iron salts form precipitates of Prussian blue granules with the hydrochloric acid, the iron solutions could be traced through this reaction into various structures of the labyrinth. From their distribution certain conclusions were drawn regarding the existence of currents within the labyrinthine fluids and possible places of resorption of endo- and perilymph.

This paper reports the results of further experiments in rabbits, cats and monkeys.

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 27, 1950.

†From the Department of Otolaryngology, Columbia University, College of Physicians and Surgeons, New York, N. Y.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

EXPERIMENTS ON RABBITS.

In 15 rabbits, after withdrawal of some spinal fluid, 0.2 to 1 cc. of a 1 per cent or 5 per cent iron-salt solution was injected into the cerebellomedullar cisterna. In view of the possibility of a rapid resorption of the salts within the cochlea, the animals were killed one to 15 minutes after the injection.

After injection of the 1 per cent solution, the salts could be detected after six minutes in the cochlea in only one rabbit out of five, and this to a very limited degree; after injection of the 5 per cent solution, the salts were found in the cochlea in five rabbits out of seven. The most important factor in the extent and speed of penetration seems, therefore, the concentration of the solution.

The distribution of the granules within the various structures of the cochlea indicates a typical pattern for the spread of the salts. Once they have entered the scala tympani they can be traced upward to the helicotrema and from there downward into the scala vestibuli.

Only a part of the salts which enter the scala tympani, however, passes upward towards the helicotrema; another portion goes laterally into the stroma of the lower part of the spiral ligament and from there into the small venous spaces within the latter. Still another portion goes through the basilar membrane into the cochlear duct and into the various spaces between the cells of the organ of Corti. From there, the salts can be traced either in a lateral direction into the spiral ligament, mostly through the lower parts of the prominentia spiralis and the sulcus spiralis externus, or medially into the tissue and the small venous spaces of the crista spiralis or through the perineural spaces of the cochlear fibres into the spiral ganglion. From the spiral ligament precipitations of the salts can be followed, sometimes within, but mostly along the somewhat larger vessels through the interscalar septa into the modiolus. From the crista spiralis they can be traced in the veins into the modiolus. Nearly all the salts which have entered the scala vestibuli through the helicotrema penetrate either through Reissner's membrane into the cochlear duct or into the uppermost part of the spiral ligament or into the crista spiralis. In 15 minutes only a very small part of the salts reaches the perilymphatic spaces of the vestibule. Here they can be traced into the lumen of the trabecular vessels. All the salts which have entered the cochlear duct within the same period of time are resorbed there and none have passed through the reunient duct into the saccule or beyond. In five rabbits granules were found in the medial (inner) wall of the endolymphatic sac. Their presence is most probably the result of an intravital penetration from the subarachnoid space through a circumscribed area of the adjacent dural wall along a projection of the dura.

In those cases in which only smaller amounts of the salts have entered the scala tympani, they are resorbed either into the spiral ligament on the lateral wall alone or through the basilar membrane into the cochlear duct of the first and successive portions of the second turn. The findings indicate that the resorptive mechanism within the cochlea is highly effective and able to cope with even relatively large amounts of iron salts. From the fundus of the internal auditory meatus the salts extend along the perineural spaces of the cochlear nerve into the spiral ganglion of the basal turn and into the stroma of the maculae and cristae. This penetration is, however, never extensive and always insignificant in comparison with the penetration through the cochlear aqueduct.

These findings in the rabbits confirm to a large extent the observations reported in our preliminary report. An important new finding seems the demonstration of a spread of the salts from the subarachnoid space through a circumscribed area of the dura into the tissue of the endolymphatic sac and from there into some of the blood vessels. Granules found in this area, or even in the lumen of the sac, are not, therefore, as previously assumed, conclusive evidence of a current directed from the cochlea into the sac. The penetration of granules from the perilymphatic spaces into the lumen of some of the trabecular vessels deserves great interest because it indicates resorption of the perilymph in these areas. Furthermore, it was demonstrated that the spread of the salts through the interscalar septa into the modiolus occurs primarily not within the blood vessels but in the perivascular spaces along the vessels.

EXPERIMENTS ON CATS.

In a second series of experiments 0.1 cc. of a 1 per cent solution was injected in four cats directly into the scala tympani through the round window membrane after exposure and opening of the bulla tympanica. The injections were made on both sides, but a few minutes apart. The animals were killed five to 16 minutes after the injection.

In each of the cats a far larger amount of the solution entered the scala tympani than in any of the rabbits. In this way a heavy overloading of the resorptive mechanisms of the cochlea was effected. A part of the iron salts injected into the scala tympani went into the aqueductus cochleae and emerged from there into the subarachnoid space. This "reverse flow" is a manifestation of a simple diffusion of the salts or the result of an increased pressure within the scala during the injection which forces the fluid in this direction.

The major portion of the injected fluid, however, spreads within the cochlea in the same directions as in rabbits injected intrathecally. Although the amounts of iron salts which reach the perilymphatic cisterna of the vestibule are larger than in rabbits, they are still comparatively small, a fact which again demonstrates the effectiveness of the resorptive mechanism of the cochlea. Similarly, only a small amount of the salts which have passed from the scalae into the cochlear duct is not resorbed from the duct, and enters the saccule through the reunient duct.

The salts which have penetrated into the perilymphatic spaces of the vestibule from the scala vestibuli might spread from there into the perilymphatic spaces of the semicircular canals. They might enter the lumen of some of the small veins in the trabecula of the perilymphatic tissue or penetrate, mainly along the perivascular spaces, through the inner wall of the vestibule into the arachnoid spaces of the fundus of the internal auditory meatus. Within the perilymphatic spaces themselves larger accumulations of salts were found, particularly on the outer surface of the walls of the maculae of the saccule and utricle and of the cristae ampullares, and also around the nerves which run from the vestibular or ampullar wall to these structures. These accumulations indicate the presence of movements of the salts to these areas. The extension of granules from these locations along the nerve fibres into the stroma of the maculae, and to a lesser extent also of the cristae and also in the opposite direction, along the nerve fibres and sometimes in the lumen of small vessels towards Scarpa's ganglion and the arachnoid meshwork of the fundus, indicates a subsequent spread into the opposite directions. In some instances the salts have evidently passed from the stroma of the maculae and cristae into the lumen of the endolymphatic spaces. There is also some evidence of a penetration of the salts through other parts of the walls of the membranous labyrinth into the lumen.

The salts which entered the saccule could be traced from there through the saccular duct into the endolymphatic duct and sac, but in none of these cases was a larger number of granules found in these cavities and in none was there evidence of resorption of the salts from the sac into the perisaccular tissue within 16 minutes after injection into the scala tympani. The precipitated granules in the lumen of the utricle and the semicircular canals seem to have come exclusively from the perilymphatic spaces by way of penetration along the nerve fibres at the indicated areas or directly through the membranous walls.

EXPERIMENTS ON MONKEYS.

In a third series of experiments nine Rhesus monkeys were studied.

In one instance only nine minutes after the intrathecal injection granules were found within the cochlear aqueducts and within the labyrinthine cavities. Within the cochlea they showed approximately the same distribution as in the rabbits and the cats. Other granules could be traced from the fundus of the internal auditory meatus along the perineural spaces of the saccular and vestibular nerves into the respective maculae. There were no granules in the lumen of the lowest part of the cochlear duct, the saccule or the saccular duct. Numerous granules were found, however, in the tissue spaces and the small blood vessels of the perisaccular tissue surrounding the rugose portion of the endolymphatic sac becoming less numerous in the distal part of the sac. The granules decreased in intensity towards the lumen, but some were found there, sometimes within the phagocytes. It would be hardly conceivable that within nine minutes all these granules should be carried through an endolymphatic current all the way down from the cochlear duct without leaving any traces in the intervening parts. Penetration of the solution from the subarachnoid space through a circumscribed area of the dura into the perisaccular tissue and into the blood vessels, and to a limited extent into the lumen, similar to the movement of the salts described in the rabbits, seems more probable.

The greater length and the narrowness of the aqueductus cochleae in monkeys seem mainly responsible for the fact that in only one case out of nine the granules had penetrated into the cochlea.

DISCUSSION.

The spread of the injected iron salts in the rabbits and monkeys from the cerebellomedullar cisterna into the fundus of the internal auditory meatus and the cochlear aqueduct could be the result of the currents within the cerebrospinal fluid together with the action of all the physicochemical factors governing the distribution of dissolved substances within fluids. Biological membranes encountered by the dissolved iron salts could modify but not materially change the spread towards the inner ear. The assumption of a special fluid current directed from the subarachnoid space towards the inner ear seems unnecessary. Such a fluid current has, however, been postulated because certain insoluble substances, such as

India ink or animal charcoal, introduced into the subarachnoid space might also penetrate into the inner ear. Sometimes in man, blood is found in the scala tympani following a hemorrhage into the subarachnoid space. A spread of these substances into the aqueductus cochleae, which is in a certain sense only a recess of the subarachnoid space, cannot be taken, however, as convincing proof for the existence of such a fluid current. Since the particles in question are very small, their spread into all parts of the subarachnoid space, their penetration into the scala tympani, and their passage from the fundus of the internal auditory meatus into the modiolus or other parts of the inner ear could again be the result of the physicochemical factors governing the distribution of small particles.

Although the possibility that a certain small proportion of the perilymph originates from the cerebrospinal fluid and enters via the cochlear aqueduct and perhaps also through the internal auditory meatus cannot be definitely denied, the cerebrospinal fluid does not seem an essential source of the perilymph. Most probably there is only very little, if any, actual fluid exchange between the subarachnoid space and the labyrinthine cavities.

The distribution of the iron salts within the various structures of the cochlea cannot be explained solely by the action of physicochemical factors. The accumulation of granules in certain areas of the spiral ligament and in the crista spiralis and the relative freedom from granules of other areas, such as the stria vascularis, indicates the action of specific biological factors. It points to a resorption of the iron salts in certain areas into the tissue spaces. From there the salts can be traced into blood vessels or into fluid spaces which surround the modiolar vessels and from the latter into the fundus of the internal auditory meatus. The main resorption of the iron salts seems to take place from the cochlear duct, and only to a lesser extent directly through the scalae. This is facilitated by the free permeability of the basilar membrane and Reissner's membrane for the salts. Since salts are only resorbed in dissolved form together with fluids, fluid movements must exist, directed in the scalae towards the lower and upper parts of the spiral ligament and in the cochlear duct towards the external spiral sulcus, the spiral ligament and the crista spiralis. The spread of the salts within the scala tympani upward to the helicotrema and from there downward through the scala vestibuli is, therefore, at least partly controlled by the actual fluid movements within the scalae and by the fact that a large-scale penetration through the basilar membrane and Reissner's membrane takes place. The biological forces behind these fluid movements are similar to those controlling the production and resorption of the cerebrospinal fluid.

A partial confirmation of our views is furnished by the histological findings of Saxen according to whom desquamated and degenerated epithelial cells and other debris in the cochlear duct in dogs and man are removed through the resorptive action of the epithelium of the sulcus spiralis externus. The extent to which endolymphatic fluid is also removed in the same way could not be found out by the histologic methods employed by Saxen. That this resorption of fluid actually takes place is proved, however, by our experiments.

The conclusion arrived at by us, that a very active resorption takes place in the cochlear duct, does not contradict the existence of a fluid current within the cochlear duct directed towards the endolymphatic duct and sac as indicated by the experiments of Guild.

The results of our experiments, however, seem to demonstrate quite clearly that the current from the cochlea to the endolymphatic sac is without great significance for the resorption of low molecular substances in physiological concentrations from the cochlea. The latter will be disposed of by the resorptive mechanism within the cochlear duct. Only when excessive amounts penetrate into the cochlear duct, as in Guild's guinea pigs or in our cats, will they be carried down to the sac. The metabolites resulting from the function of the cells of the organ of Corti which penetrate into the endolymph are evidently also removed by this local resorptive mechanism. It would be hard to understand why, for instance, metabolites originating in the apical turn of the cochlea should be carried all the way down through the cochlear duct into the saccule and thereby come in contact with other parts of the organ of Corti.

The resorption from the cochlear duct of high molecular substances, of emulsions of fats, and of corpuscular elements is perhaps less efficient than that of the low molescular substances used in our experiments. It is conceivable that larger proportions of them might reach the endolymphatic sac.

That portion of the salts which was not resorbed from the scalae eventually reaches the vestibule, particularly when their concentration in the perilymph was as great as in our cats injected through the round window. In these instances, the iron salts had also spread from the scala tympani through the cochlear aqueduct into the subarachnoid space. This would have been difficult to explain if a strong fluid current from the subarachnoid space into the scala tympani had really existed as assumed by many authors; however, it becomes easily understandable if one assumes that there is no fluid current, or hardly any, within the cochlear aqueduct, and that salts can spread through the latter in either direction, depending upon their relative concentration in the cerebrospinal fluid and in the labyrinthine fluids.

The fate of the salts which have gotten into the vestibule has already been discussed in the cat experiments.

The spread from the vestibule to the fundus of the internal auditory meatus gives rise to the same questions as the spread from the scala tympani to the subarachnoid space. It would be incompatible with the existence of a strong fluid current from the fundus into the labyrinth, and one comes to the conclusion again that such a current does not exist and that a spread might occur in either direction, depending upon the relative concentration of the salts in the fundus and the vestibule.

Nothing definite can be said about the main source of the perilymphatic fluid. It seems much more probable that it is derived from the perilymphatic blood vessels than that it originates from the endolymph and passes through the walls of the membranous labyrinth. The walls of the membranous labyrinth seem to be easily penetrated by low-molecular substances, and this could explain why the peri- and endolymph are virtually isosmotic. The fact that in our experiments the iron salts moved from the peri- into the endolymphatic space simply demonstrates the high degree of permeability of the membranes and should not be taken as an indication that this is the prevailing direction of movement of dissolved substances under physiological conditions. Normally it is more likely to be from the endolymph towards the perilymph.

The resorption of the cochlear perilymph seems to take place from both scalae into the tissue spaces and from there into the blood vessels of the spiral ligament, from the vestibule and the semicircular canals into the blood vessels of the trabecula of the perilymphatic tissue. The spread into the perivascular and perineural spaces is probably of lesser importance under physiological conditions. Both spaces are more likely to represent auxiliary avenues of escape for dissolved substances if their concentration is so high that the normal pathways have become overloaded.

The salts which have passed into the lumen of the saccule can be traced from there into the endolymphatic duct and sac. Because resorption of endolymph takes place in certain areas of the endolymphatic sac, the salts have probably been carried down to these areas not solely by diffusion but also by an actual fluid current. That no evidence of resorption from the lumen of the sac was seen in any of our specimens could have been due to the relatively short time interval between the injection of the salts and the death of the animals.

Great caution is advisable in the interpretation of the findings in and around the endolymphatic sac because of diffusion of the salts from a circumscribed area of the subarachnoid space along a projection of the dura into the perisaccular tissue. Here the presence of granules could easily be mistaken as evidence of resorption from the lumen or of transudation from the blood stream. There was nothing in the distribution of the granules in our specimens which could be interpreted as an indication of a current from the utricle to the endolymphatic sac.

The possibility of a local resorption of endolymph from the lumen of the saccule and utricle into the stroma of the maculae and from the ampullae into the stroma of the cristae must be considered, although the histologic findings in the examined cases do not permit any definite conclusions regarding these questions.

SUMMARY AND CONCLUSIONS.

Using the method of Weed, iron salt solutions of various concentrations were injected into the subarachnoid space of 15 rabbits and nine Rhesus monkeys. Similarly, in four cats iron salts were injected through the round window membrane directly into the scala tympani.

The iron salts spread readily through the wide aqueductus cochleae of the rabbits and less readily through the narrow aqueduct of the monkeys into the scala tympani. Another less extensive spread may occur from the fundus of the internal auditory meatus mainly in the perineural spaces of the respective nerves into the modiolus, the maculae and the cristae ampullares. Membranes encountered by the iron salts do not seem to slow up this spread perceptibly. In the cats the iron salts spread in the opposite direction; from the scala tympani through the aqueduct into the subarachnoid space, from the modiolus, the vestibule and the perilymphatic spaces of the semicircular canals into the fundus of the internal auditory meatus.

These findings are regarded as an indication that active fluid currents are absent in either direction, and that the spread of the iron salts is mainly or entirely the result of the action of the usual physicochemical factors governing the distribution of dissolved substances within fluids.

In the scala tympani the salts spread partly upward to the helicotrema and partly through the basilar membrane into the cochlear duct, while another part was resorbed into the spiral ligament. From the helicotrema the salts spread in the scala vestibuli towards the vestibule. A part of them passed through Reissner's membrane into the cochlear duct, and another part was resorbed directly into the spiral ligament. If sufficient amounts of iron salts reach the vestibule, they spread from there into the perilymphatic spaces of the semicircular canals and eventually into the blood vessels of the trabecula of the perilymphatic tissue. Other portions pass through the walls of the membranous labyrinth into the endolymphatic space. If the concentration of the salts in the perilymph is very high, they may spread along the perineural and perivascular spaces in either direction towards the maculae and cristae and into the fundus of the internal auditory meatus. The findings indicate that actual resorption of perilymphatic fluid takes place mainly in three areas: from both scalae into the spiral ligament and from the vestibule and the semicircular canals into the blood vessels in the trabecula of the perilymphatic tissue.

Since no perceptible fluid current towards the perilymphatic space could be demonstrated, neither from the subarachnoid nor from the endolymphatic space, at least the main part of the perilymphatic fluid is evidently formed in the perilymphatic space itself, most likely from the blood vessels. The marked permeability of the walls of the endolymphatic system makes it probable that a spread of dissolved substances takes place from the endo- into the perilymphatic fluid or vice versa.

Within the cochlear duct resorption of iron salts occurs through the sulcus spiralis externus and the spiral ligament, and through the crista spiralis, indicating the resorption of endolymph in the same areas. The resorptive mechanism of the cochlea seems more than sufficient for the disposal of low molecular substances in physiological concentrations.

Resorption of the endolymph from a certain part of the endolymphatic sac, although not visible in the specimens, seems an established fact and indicates the presence of an evidently slow fluid current towards the sac. This current seems to affect mainly those substances which are difficult for resorption by the regular cochlear mechanism and are carried into the sac. Presence of granules in the subepithelial tissue of the endolymphatic sac is, however, not necessarily an evidence of resorption from the lumen because of diffusion of the salts from a circumscribed area of the subarachnoid space along a projection of the dura into the perisaccular tissue.

MODERN TEMPORAL BONE SURGERY — HISTORY OF ITS EVOLUTION.[*]

JULIUS LEMPERT, M.D.,[†][‡]
New York, N. Y.

Thirty years ago when I began to practice otology I was not quite satisfied with the results that I personally obtained in temporal bone surgery, and on observing the results of others I was not more satisfied with theirs. Especially disconcerting to me at that time were the extremely poor results obtained with the traditionally accepted and employed surgical treatment of obstructive exostosis of the external auditory canal.

When exostosis of the external auditory canal becomes sufficiently obstructive to interfere with visualization of the tympanic membrane and to interfere with transmission of sound to the middle ear, or with drainage of an acute or chronic middle ear infection, surgical removal of the bony mass becomes essential, whatever its size, extent or location may be.

The accepted and most commonly employed surgical procedure for many years for the cure of such a condition consisted of enlarging the meatus through a postauricular incision, by removing the posterior bony wall of the external auditory canal and making a conchal flap, as in the postauricular radical mastoidectomy, and lining the mastoid cavity with skin grafts. The operation involved too much trauma and too much unnecessary destruction of healthy tissue for the removal of an innocent mass of bone from within the external auditory canal. The consequences in many of these cases were compli-

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[*] Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 26, 1950.

[†] Lempert Institute of Otology.

[‡] Lempert Research Foundation, Inc.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 7, 1950.

cations, sometimes intracranial. It all seemed very unnecessary to me, and I decided to look for and find a way of surgically removing an exostosis lesion from the external auditory canal without opening and destroying the mastoid process.

That decision marked the beginning of my endaural surgery. I operated upon my next case of exostosis as follows: I made an incision along the anterior margin of the concha down to the bony mastoid cortex, then subperiosteally elevated the skin and periosteum covering the exostosis mass with a narrow periosteal elevator slowly, a few millimeters at a time. This elevation was done slowly because the obstructive bony mass is as a rule irregular in shape and contour, and if an attempt is made to separate the cutaneous lining, which becomes thinner and thinner as it approaches the hidden tympanic membrane, it may be injured and torn; therefore, I separated a few millimeters at a time. With a small electrically driven polishing burr I then removed as much of the exostosis mass from the posterior, inferoposterior and superoposterior walls as was exposed to view. This having been done, I continued my elevation of the skin and periosteum a few millimeters at a time and gradually removed the exostosis mass wherever found down to the anterior mastoid cortex without opening any of the mastoid cells until the tympanic ring was reached. After removing the entire bony mass I replaced the skin and periosteum into its normal position against the anterior mastoid cortex. The entire circumference of the tympanic membrane became visible and the operation was over.

I then packed the canal with paraffin mesh gauze, removing it at the end of 48 hours. Incision is sutured. Primary union is effected. This is the complete procedure. The many men who have since tried this procedure of mine would never again think of opening a mastoid process to remove an exostosis from the external auditory canal. I have employed this method ever since and found it to be the most satisfactory procedure for the otologist and for the patient. It can be done under local or general anesthesia and hospitalization need not be more than one or two days (see Figs. 1-5).

ENDAURAL REMOVAL OF OBSTRUCTIVE EXOSTOSIS FROM EXTERNAL AUDITORY CANAL.

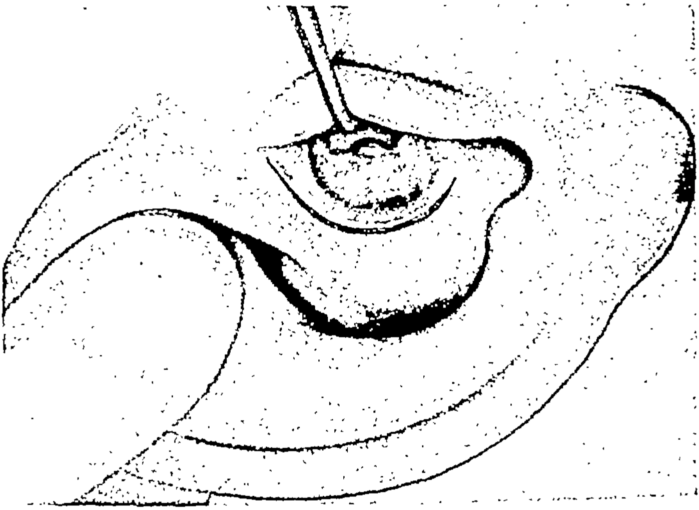

Fig. 1. Step I. Endaural incision along anterior margin of concha.

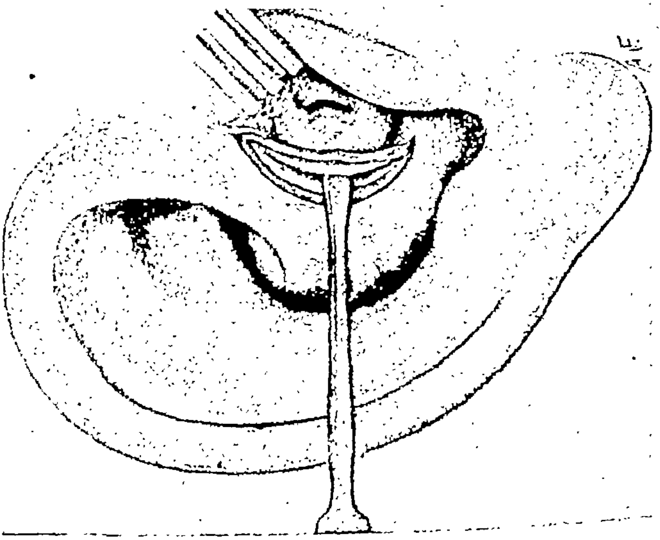

Fig. 2. Step II. Subperiosteal elevation of skin and periosteum from region of exostosis mass.

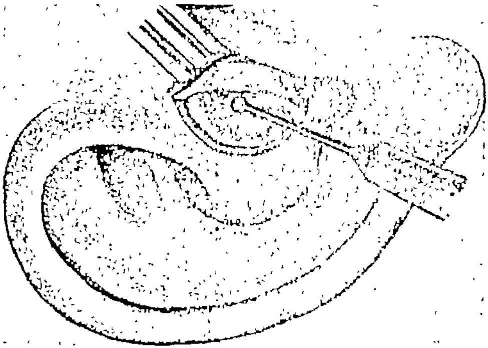

Fig. 3. Step III. Exostosis mass is removed with electrically driven burr.

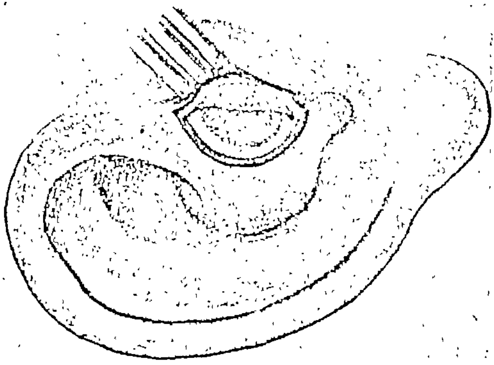

Fig. 1. Step IV. Exostosis mass was removed.

POSTAURICULAR FISTULA.

One of the commonly met conditions in those days and which confronts us occasionally today, calling for surgical correction by the otologist, was the permanently open postauricular fistula. Such a fistula was either *1.* the result of arrested and incomplete healing of the postauricular mastoid wound following the simple mastoidectomy when the postauricular incision was left open to maintain drainage of the infection; *2.* the repeated infection and breakdown of the healed postauricular scar following the simple mastoidectomy because of repeated recurrence of acute attacks of otitis media, or *3.* the sloughing and breakdown of the freshly sutured postauricular mastoid incision following the postauricular radical mastoidectomy.

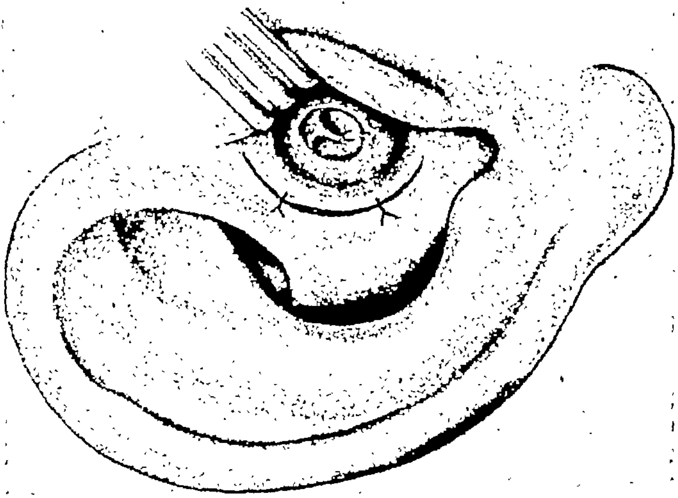

Fig. 5. Step V. Normal contour of external auditory canal has been re-established. The tympanic membrane is now visible. The subperiosteally elevated skin and periosteum is replaced and held in position with paraffin mesh gauze.

Very many methods of surgically closing such a postauricular fistula were variously employed by otologists. The underlying surgical principle of all these methods was the borrowing and diverting of skin, periosteum and also muscle from the neighborhood of the postauricular fistula for the correction and closure of the fistular defect. The results obtained following these surgical efforts were not satisfactory as a rule and I thought that there was something wrong and that there should be a means of permanently closing such a fistula without sacrificing and deforming the healthy tissue around it. After observing that a postauricular fistula is always either completely or partially lined with a funnel-shaped invagination of skin and periosteum, I decided to proceed as follows, again endaurally:

I made an endaural incision along the anterior margin of the concha, down to the mastoid cortex through the skin and periosteum, and with a narrow periosteal elevator, I separated and elevated the auricle and postauricular skin and periosteum subperiosteally in a posterior direction up to the margin of the outer and wider circumference of the funnel-shaped invagination of skin and periosteum lining the postauricular fistula. The intention being to mobilize the auricle so that I could freely move it in any direction I wished. By slowly and gently elevating the inwardly adherent skin and periosteum lining the fistula through the eudaural incision with the narrow periosteal elevator, I everted outward the funnel of skin and periosteum. By converting the inwardly directed funnel of skin and periosteum into an outwardly directed funnel without severing it from its continuity with the postauricular skin and periosteum I was confronted with more skin and periosteum than needed for closing the postauricular fistula. If there are any infected cells — if an incomplete mastoidectomy had been done and there is any infection within that region — I remove the pathologic processes. With fine scissors I removed the excessive skin and periosteum and then sutured the freshly cut edges of skin and periosteum. I put a little rubber tissue drain into the original incision made endaurally, which was removed at the end of five days. A perfectly endaural and postaural wound was the result. This can be done under local or general anesthesia. The procedure is being carried out with the greatest of satisfaction by many men all over the world today (see Figs. 6-12).

COMPLETE ENDAURAL SUBCORTICAL MASTOIDOTYMPANECTOMY.

The most commonly encountered postauricular fistulas in those days were secondary to the performance of the postauricular radical mastoidectomy, and these were the most difficult to correct. While correcting these fistulas I could not

LEMPERT PLASTIC FOR CLOSURE OF POSTAURICULAR FISTULA.

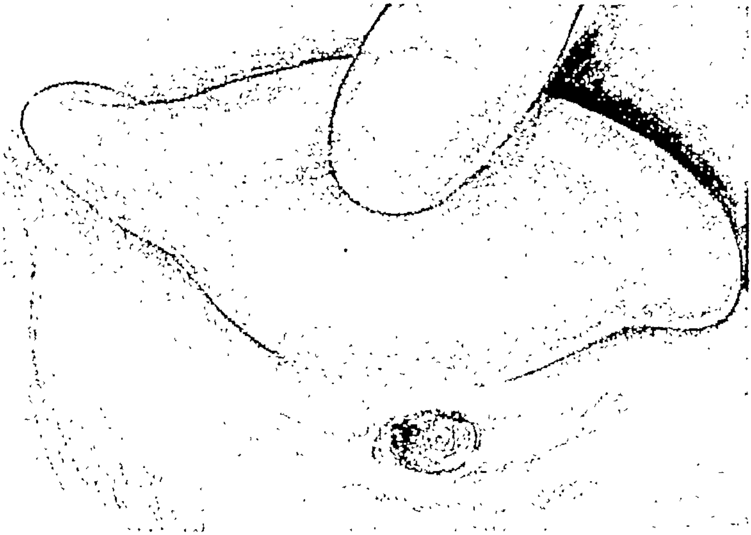

Fig. 6. Postauricular fistula.

help thinking, "Why do we get fistulas following the radical mastoidectomy?" "Couldn't a means of performing a radical

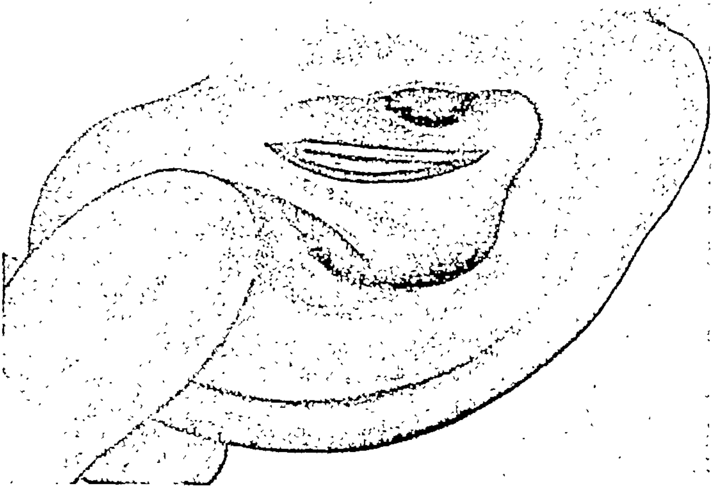

Fig. 7. Step I. Endaural incision through skin and periosteum is made along anterior margin of concha.

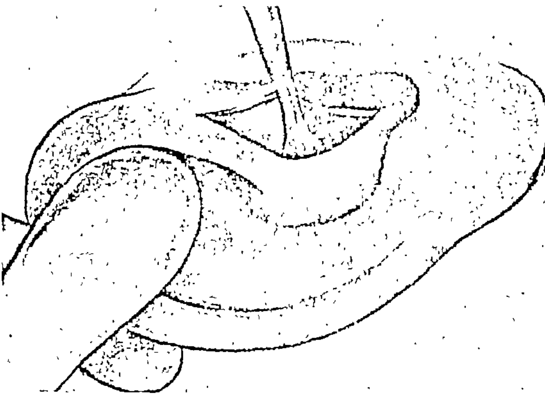

Fig. 8. Step II. Subperiosteal elevation of auricle from its bony attachments through endaural incision.

mastoidectomy be devised which would render the occurrence of a postauricular fistula impossible?"

On analyzing the whys and wherefores of fistula following the radical mastoidectomy it seemed natural that a fistula should occur when one considered the technique of performing a postauricular radical mastoidectomy. First of all we make a postauricular incision in order to open the mastoid antrum and to exenterate the pathology from the mastoid process. Can we do a radical mastoidectomy which necessitates also the removal of the pathologic processes from within the tympanic cavity, through a postauricular incision? We cannot, and that is the reason why we must continue to destroy and remove the posterior bony wall of the external auditory canal plus the skin and periosteum covering it, in order to expose to view the tympanic cavity and render it accessible for surgical intervention. In other words, when doing a radical mastoidectomy postauricularly, we cannot reach the tympanic cavity surgically without destroying also the posterior wall of the external auditory canal; we cannot

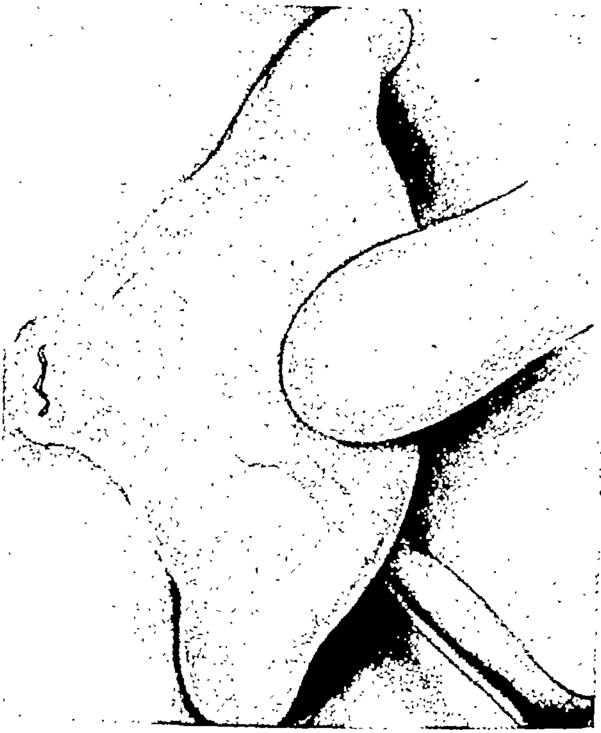

Fig. 9. Step III. Subperiosteal elevation and eversion outward of inverted skin lining postauricular fistula through the endaural incision. The postauricular skin deficiency is thus converted into a skin surplus.

reach it through the postauricular approach unless we supplement it with an endaural approach. To speak of the Schwartze radical mastoidectomy as a postauricular technique is really a misrepresentation of the facts, since in reality it is a combined postauricular and endaural technique. It became quite obvious to me that a technique which involves such wholesale destruction of soft tissue surrounding the auricle will undermine and weaken the whole structural foundation of the auricle, and as a result it is quite natural in the presence of infection for the sutured postauricular incision to become infected and to open up with a resulting permanent postauricular fistula.

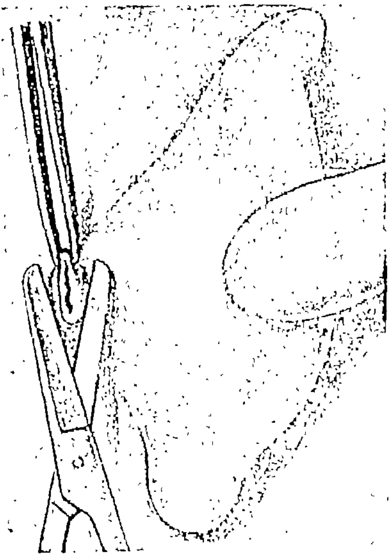

Fig. 10. Step IV. Removal of everted skin surplus.

It thus became quite obvious to me at the time that if I could do a radical mastoidectomy without a postauricular incision, the occurrence of a postauricular fistula would no longer be possible. I decided to try to do the radical mastoidectomy endaurally only and without a postauricular incision. After trying various endaural incisions I finally concluded that the best endaural approach for a radical mastoidectomy requires the following four endaural incisions: The first incision is made along the posterior margin of the tympanic ring through skin and periosteum; the second one is made along the entire superoposterior wall of the external auditory canal coming right out of the canal up to the apex of the supramastoid cortex, or by attempting to remove the pathology covering and hiding from view the vital anatomy within the tympanic spaces. His constant fear of injuring the vital anatomy was due to the fact that in performing the Schwartze postauricular radical mastoidectomy he had no other safeguard except the hope of avoiding injury to the anatomy which most often he could not see. These same mental reservations which prompted him not to promise the patient a dry ear should also have prevented his suggesting that the possible onset of an intracranial complication could thus be prevented. Even the extremely skillful otologist, after extensive experience with the postauricular radical mastoidectomy technique, will not infrequently encounter difficulties beyond his expectation. Being fully cognizant of the inherent difficulties and shortcomings of the Schwartze postauricular radical mastoidectomy technique, I have developed a simple and safe mastoidotympanectomy technique. Any relatively young in experience, well-trained otologist can employ this technique without fear of injuring vital anatomy and with full confidence in his ability to remove all the pathologic processes responsible for the persistent middle ear discharge. The electric headlight and the magnifying loupe are invaluable assets to the safe performance of this technique.

TECHNIQUE.

The endaural subcortical mastoidotympanectomy technique is based upon the following surgical principles:

1. The abandonment of the hazardous and tedious blind search for the mastoid antrum through the outer mastoid cortex. Too many accidents occur unnecessarily as a direct result of this search.

2. The exploration of all the vital anatomy and eradication of the pathologic processes within the confines of the mastoid portion of the temporal bone by first removing the thinner anterior wall of the mastoid cortex with the electrically driven burr directed from within outward, beginning with the tympanic margin of the posterior bony wall of the external auditory canal, instead of directing the instrumentation blindly and dodgingly, first through the much thicker outer wall of the mastoid cortex towards the vital anatomy.

3. The exploration of the pathologic process within the epitympanum by engaging the tympanic margin of the superior bony wall of the external auditory canal with the electrically driven burr and removing this bony wall from within outward until the roof of the external auditory canal and the roof of the epitympanum become continuous.

4. The exploration of the pathologic process within the hypotympanum by engaging the tympanic margin of the inferior bony wall of the external auditory canal with the electrically driven burr and removing this bony wall from within outward until the floor of the hypotympanum and the floor of the external auditory canal become continuous.

5. The removal of the pathologic processes from the tympanum, epitympanum and hypotympanum which rest upon the thickened diseased mucoperiosteum lining these tympanic spaces, by elevating, separating and stripping the entire mucoperiosteum from its attachment to the tympanic bony walls and removing it together with the pathologic processes resting upon it. All the vital middle ear anatomy is thus exposed to view without predisposing it to the danger of injury. This method of removing the pathology from the middle ear spaces is analogous to the removal of the damaged rug or carpet with the dirt on it, instead of removing the dirt from the already damaged carpet.

If one hopes to obtain a dry ear as a result of this surgery, one must remove all the pathological processes encountered, any one of which when not removed could perpetuate the middle ear discharge. The removal of granulations, polypi and cholesteatomata from the mastoid process, epitympanum, tympanum and hypotympanum is not sufficient to stop a chronic middle ear discharge. For very many years there existed an infection within the middle ear, the mucoperiosteum lining, it is infected, thickened and inflamed, and one has to remove it until one exposes and inspects the bony structure underneath it. The perieustachian tube cells which are infected must be removed, but a curette must never be introduced into the Eustachian tube. The use of a curette in

LEMPERT SUBCORTICAL MASTOIDOTYMPANECTOMY.

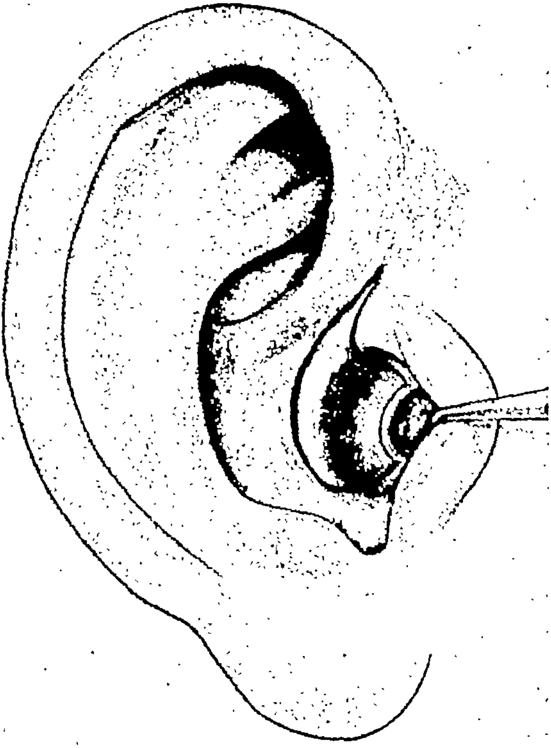

Fig. 13. Four endaural incisions are made through skin and periosteum.

the mouth of the Eustachian tube as a rule results in osteomyelitis of the bony portion of the Eustachian tube. With a No. 11 or 12 S.S. White dental excavator the granulations around the stapedial crura must be removed, and one must also remove the granulations from the round window niche, not only to remove a source of continued infection but also in the hope of giving the patient better hearing than he enjoyed before the operation. A necrotic facial canal must be removed

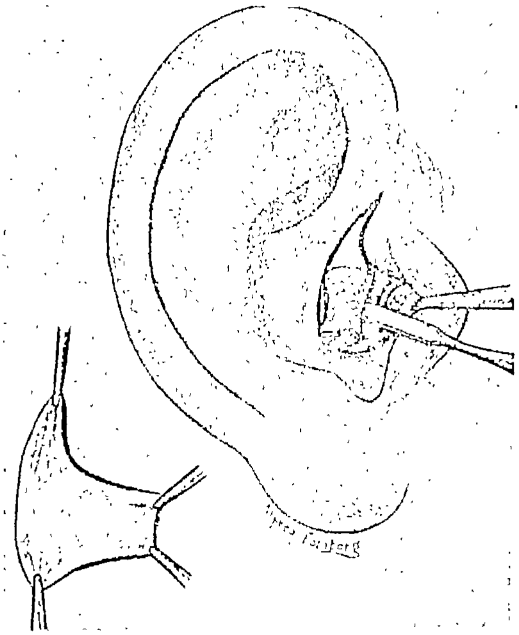

Fig. 11. Skin flap removed and saved for skin grafting postoperative bony cavity.

and the facial nerve exposed to view. A necrotic bony carotid canal must be removed and carotid artery exposed to view. Finally, if it is desired to have a dry ear the tensor tympani muscle lying within its semicanal must be removed. This muscle has been bathing in pus for very many years and a continued postoperative middle ear discharge may well result when this infected muscle is not removed. This must be done by fracturing the processus cochleariformis in a direction towards and over the facial canal, then stripping the muscle from its semicanal towards the Eustachian tube orifice and finally grasping it with a narrow pointed artery clamp and pulling it out *in toto*. The chances of obtaining a dry ear with

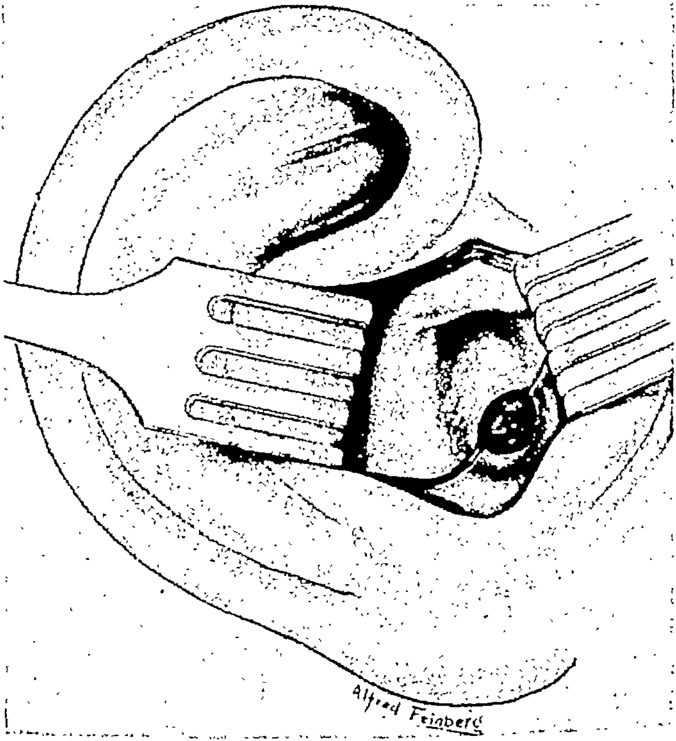

Fig. 15. The outer mastoid cortex, spina henle and the entire anterior mastoid cortex can now be seen.

the above described technique are almost 100 per cent providing the after-treatment is carried out with the same undivided attention (see Figs. 13-25).

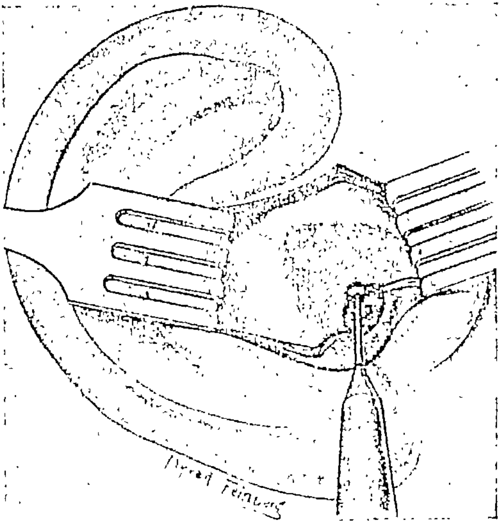

Fig. 16. The tympanic inner margin of the posterior wall of the external auditory canal is engaged with an electrically driven sharp cutting burr and the anterior wall of the mastoid cortex is gradually removed from within outward.

FACIAL NERVE INJURY REPAIR.

When temporal bone surgery is performed with proper lighting and magnification it can be done without danger of injuring the vital anatomy involved. I do not mean to say that one may not occasionally injure the facial nerve. That can happen, but when it happens we must know that it happened and how and when it happened. In those circumstances

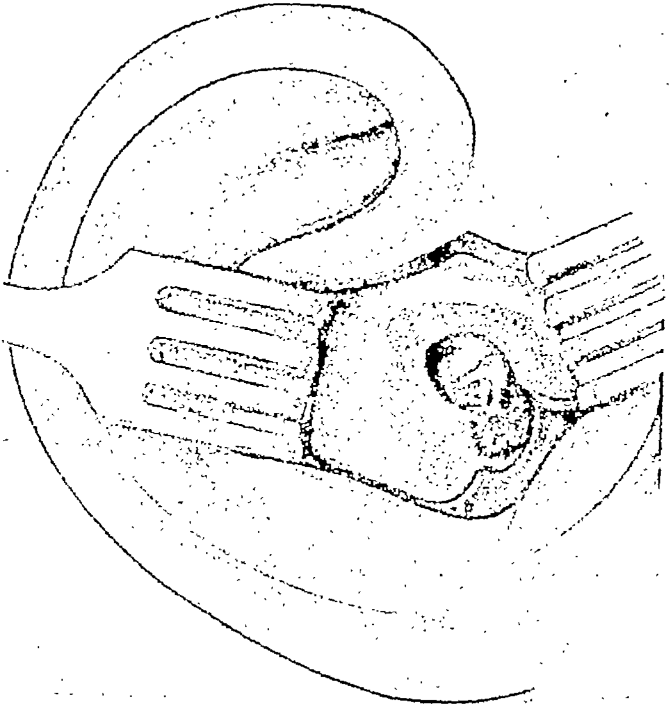

Fig. 17. The incudo stapedial joint and the external semicircular canal can be seen as result of partial removal of the anterior mastoid cortex from within outward.

we have the "right" to have an accident. But when we have an accidental injury of the facial nerve while doing a radical mastoidectomy and do not know it until the anesthetist tells us. "The face is crooked"; and we with surprise respond by saying "How did that ever happen? — I wasn't even near the facial nerve." Well, this is very sad, because the surgeon is the only one who should have known how it ever happened.

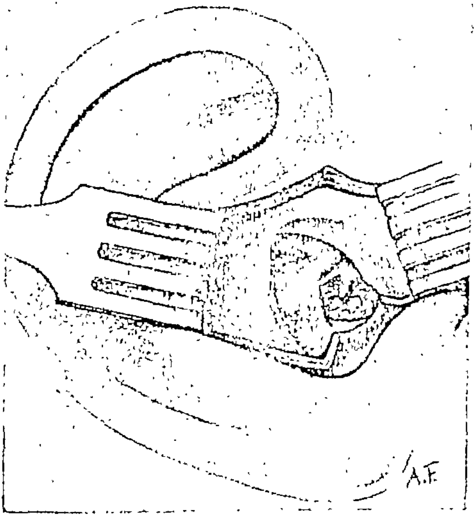

Fig. 18. Following the removal of the entire anterior wall of the mastoid cortex from within outward and the exenteration of the diseased mastoid cell structure the sharply defined semicircular canals can now be seen.

One cannot overemphasize the fact that if there is an accident we must know how it happened, where it happened, and when it happened; and if we do not know, we have no right to be doing temporal bone surgery because such a confession in the face of an obviously existing injury is concrete evidence that we never knew where we were in the temporal bone. Accidents can happen to anybody, but if I am doing this operation

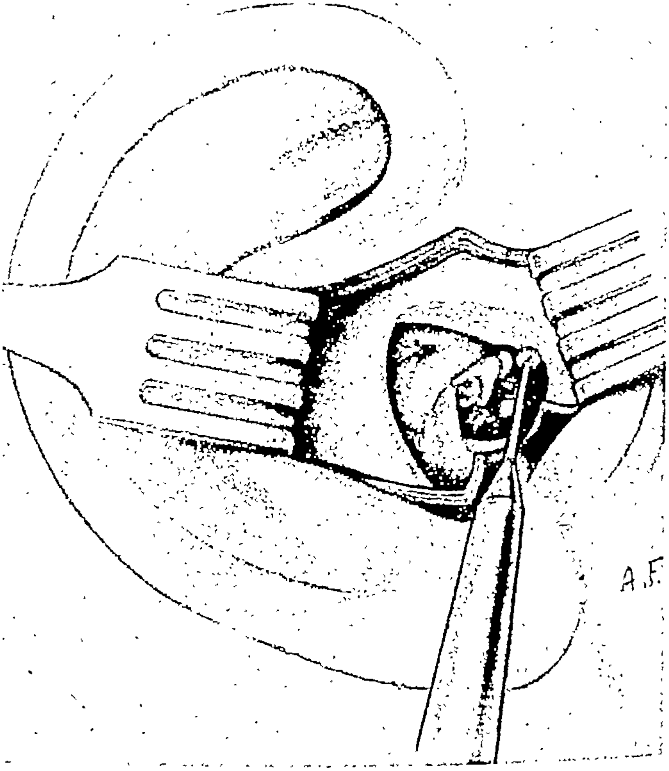

Fig. 19. To expose the pathologic process within the epitympanum the innermost margin of the superior bony wall of the external auditory canal is removed from within outward to establish a continuity between the roof of the canal and the floor of the middle fossa.

properly it is I who will tell the anesthetist, long before he tells me, "Will you look at that face? I have just injured the facial nerve." The anesthetist's response will inevitably be: "Yes! The face is crooked." The anesthetist having thus confirmed the fact that I have injured the facial nerve, my only comment to his answer is, "Thank you."

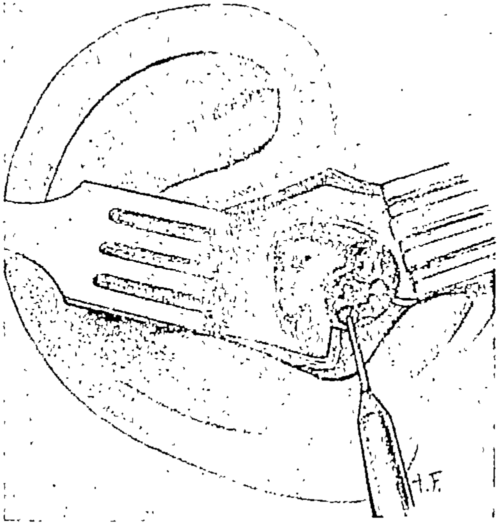

Fig. 30. To expose the pathologic process within the hypotympanum the tympanic margin of the bony floor of the external auditory canal is engaged with an electrically driven burr and removed from within outward until a continuity between the floor of the external auditory canal and the floor of the hypotympanum is established.

The most common facial nerve injury is the result of an inward fracture of bone into the Fallopian canal which compresses the facial nerve. This can be readily observed with magnifying glasses. It takes less than one minute to remove face of an obviously existing injury is concrete evidence that we never knew where we were in the temporal bone. Accidents can happen to anybody, but if I am doing this operation properly it is I who will tell the anesthetist, long before he tells me, "Will you look at that face? I have just injured the facial nerve." The anesthetist's response will inevitably be: "Yes! The face is crooked." The anesthetist having thus con-

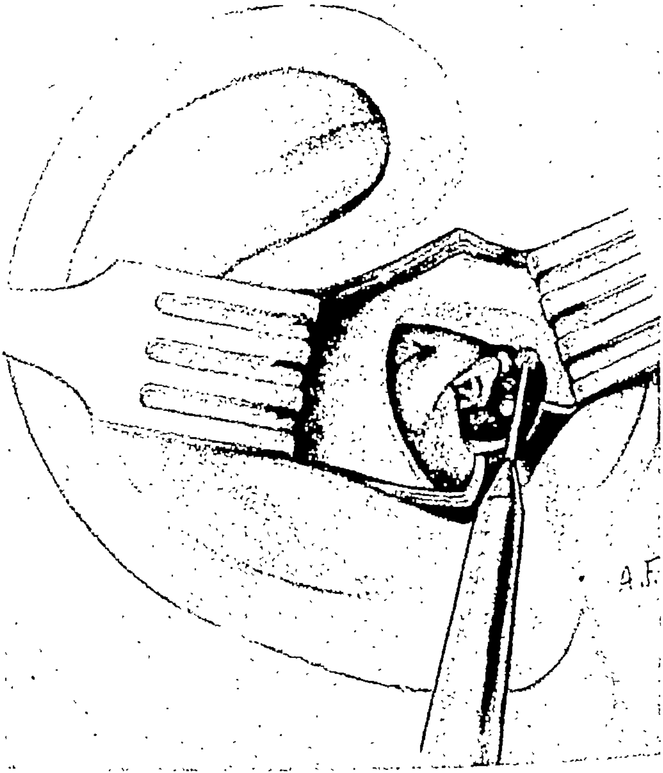

Fig. 19. To expose the pathologic process within the epitympanum the innermost margin of the superior bony wall of the external auditory canal is removed from within outward to establish a continuity between the roof of the canal and the floor of the middle fossa.

for a distance of about 5 mm. in either direction of the injury will as a rule result in healing of the nerve injury with ultimate restoration of good function; however, when the facial nerve has been torn asunder with a resulting gap between the severed ends I found that by changing the normal habitat of the nerve I could establish a favorable status for quick repair by employing the end to end anastomosis technique.

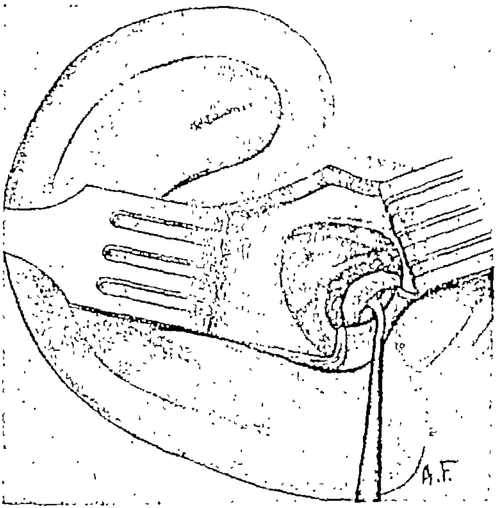

Fig. 22. The diseased mucoperiosteum together with the pathologic processes resting upon it are being removed by separating and elevating the mucoperiosteum from its attachment to the bony tympanic walls.

Since a straight line is the shortest distance between two points, I displace the facial nerve from its curved canal and convert the curved line of the nerve into a straight line. Immediately, no matter how much nerve has been missing as a result of the injury, we have much more nerve tissue left than is needed to meet end to end. After trimming both torn ends to where both meet comfortably but not too loosely to form a straight line, they are held in apposition with a fine suture.

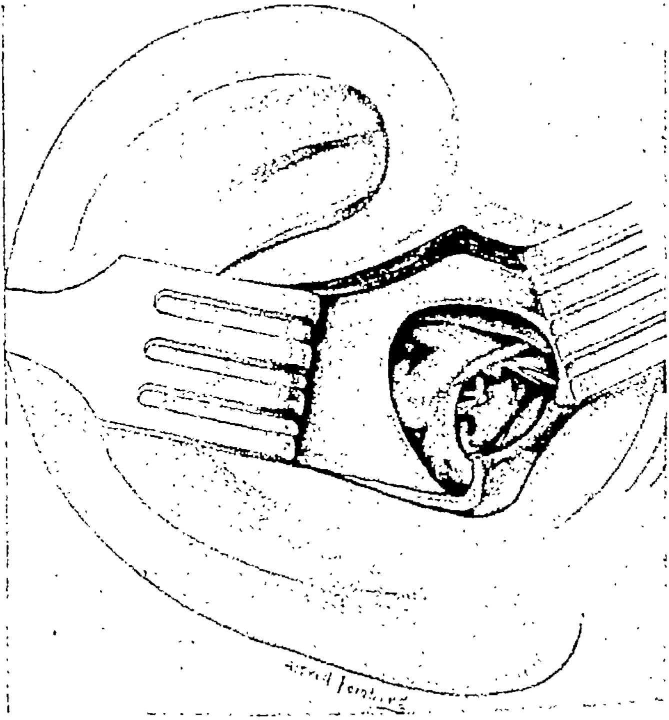

Fig. 23. The pathologic processes resting upon the mucoperiosteum having been removed, the infected tensor tympani muscle within its semicanal is now exposed to view.

End to end suture of the injured facial nerve has given me better end-results than I was able to obtain with the Ballance-Duell transplantation technique. The end to end anastomosis technique is a much simpler one than the transplantation technique. Using your magnifying glasses and proper lighting, you are the master of the situation. When I say "master of the situation" I repeat that any otologist doing temporal bone surgery must necessarily be master of the situation. He

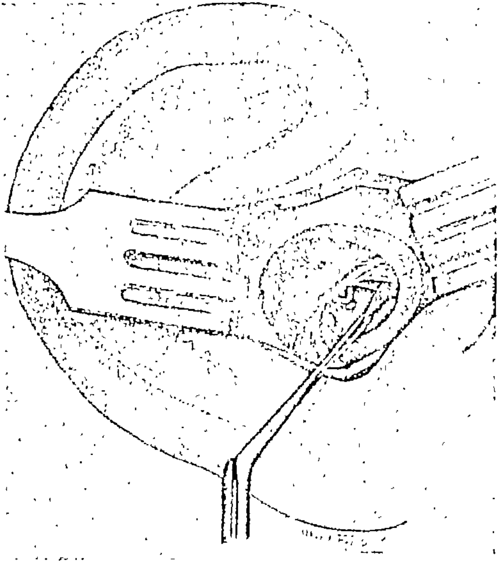

Fig. 24. The infected tensor tympani muscle is removed from its semicanal.

must be able to repair the facial nerve injury immediately on the table. He must know where, when and how he has caused any injury. The general surgeon, if he meets with an accident on the table, does not call for help and does not procrastinate; he does not permit the patient to leave the operating table, and he certainly would never think of discharging the patient from the hospital without personally repairing the injury in question, or having someone of greater skill repair it for him. He is, as a rule, the master of the situation; he immediately handles whatever accident he meets. Otologists must be trained to do the same (see Figs. 26-29).

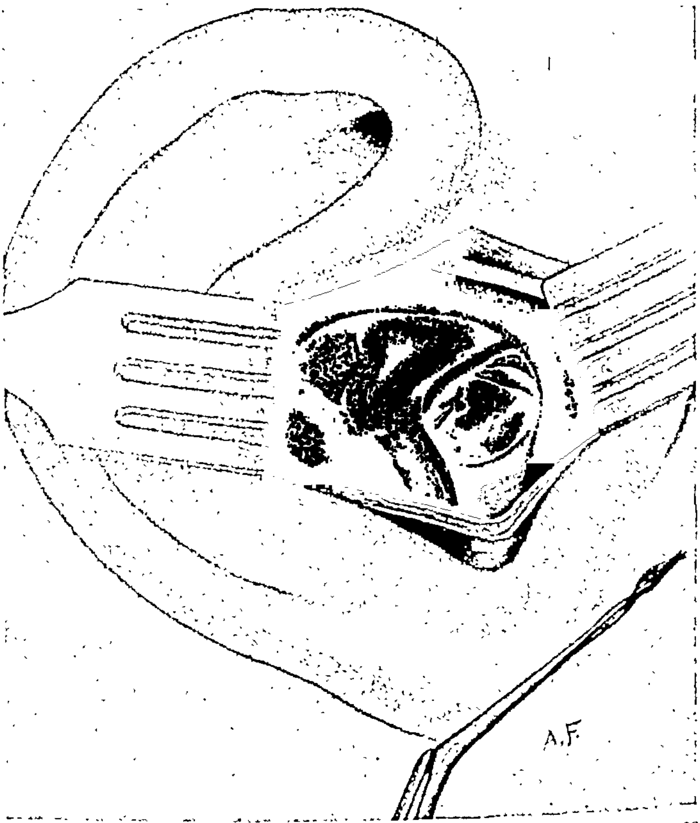

Fig. 25. The outer wall of the mastoid cortex finally removed, exposing to view all the vital anatomy within the completely exenterated mastoidotympanic wound.

LEMPERT END TO END ANASTOMOSIS OF INJURED FACIAL NERVE.

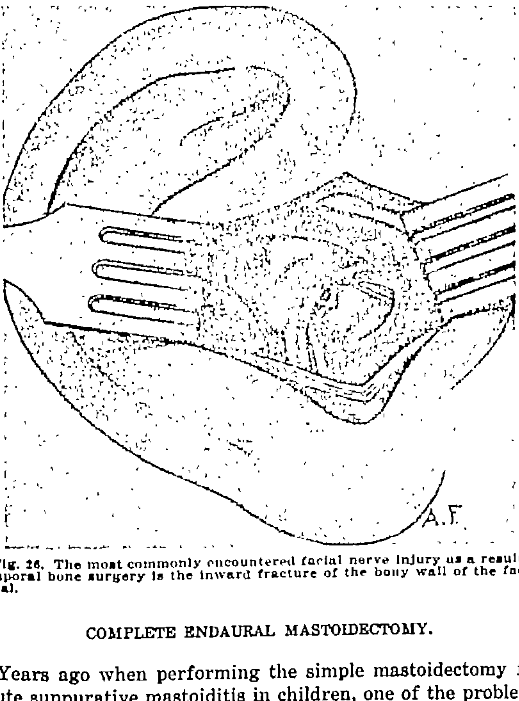

Fig. 26. The most commonly encountered facial nerve injury as a result of temporal bone surgery is the inward fracture of the bony wall of the facial canal.

COMPLETE ENDAURAL MASTOIDECTOMY.

Years ago when performing the simple mastoidectomy for acute suppurative mastoiditis in children, one of the problems which frequently haunted us was the child who was repeatedly and periodically brought back to the otologist with a ballooned-out, inflamed postauricular scar long after the postauricular mastoid wound had completely healed. This was usually the result of a newly contracted middle ear infection from varying causes. One customarily told the mother that it was just a middle ear infection, a very simple thing with secondary inflammation of the postauricular scar requiring

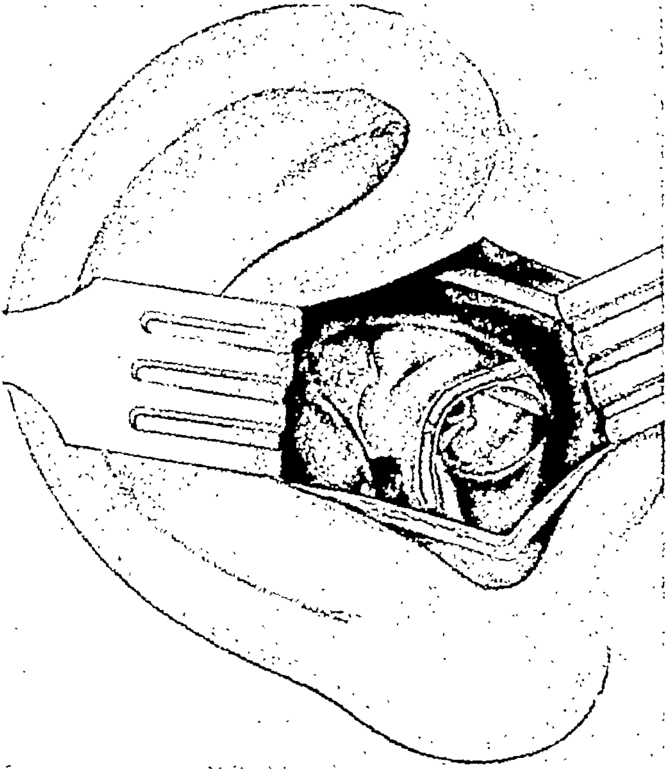

Fig. 27. Severed facial nerve is seen.

incision and drainage, and that was all. Should that patient, for some reason, have gone to one's colleague, even one's best friend, it was too often a different story. Then this inflamed ear was labeled as a recurrent mastoiditis, creating indirectly the impression that the original mastoidectomy had been incompletely performed. As a matter of fact, it has been my observation that the more completely a mastoidectomy was performed the more likely was this clinical picture to appear subsequent to a postoperatively occurring otitis media. The so-called recurrence happened only because it was the natural result. When we have a middle ear infection in the presence of a normal healthy mastoid process which was never operated upon, the course of least resistance for the infection within the middle ear is the tympanic membrane, and either

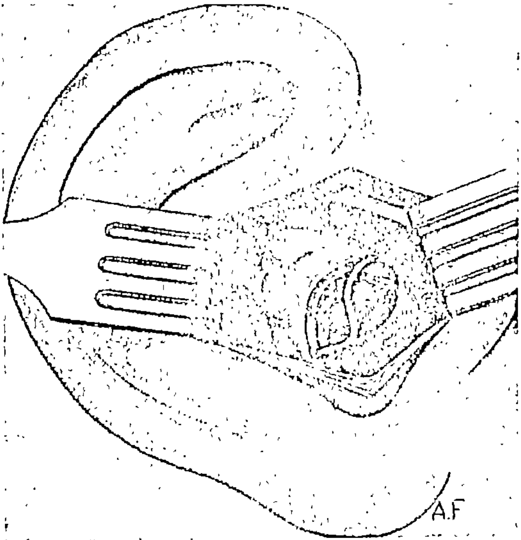

Fig. 28. The facial nerve is dislodged from its curved bed within the bony facial canal.

the membrane ruptures by itself or, if seen in time by the otologist, he incises it, drains it, and hopes for the best. When a child has had a previous mastoidectomy which thoroughly healed and then contracts a middle ear infection, the course of least resistance for such a middle ear infection is no longer the tympanic membrane. With that mastoid antrum widely open and the mastoid process widely exenterated, the infection gradually again takes the course of least resistance, which this time, however, is backward to the mastoid wound instead of forward to the tympanic membrane; and the postauricular scar tissue whose resistance is much lower than that of a healthy postauricular skin and periosteum becomes infected immediately and "balloons out." In other words, infection naturally always takes the course of least resistance.

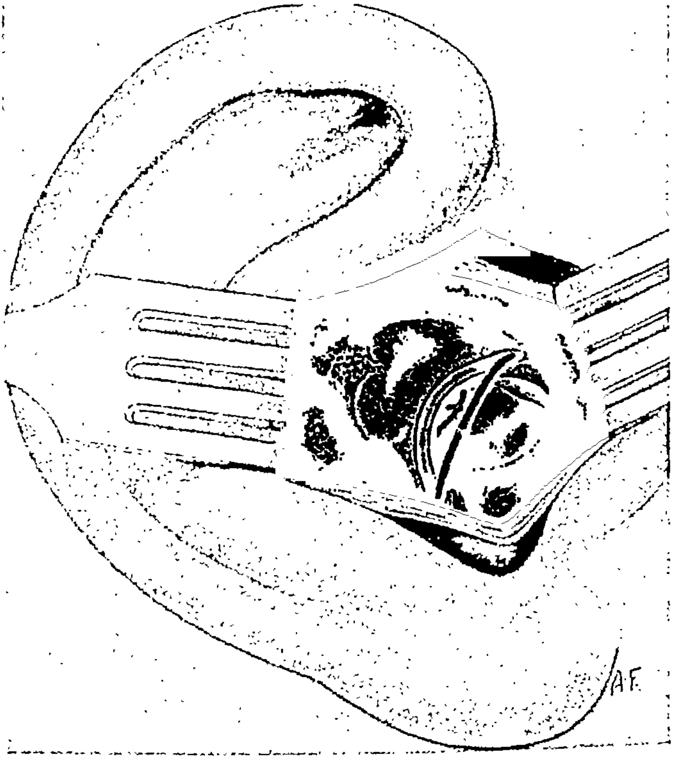

Fig. 29. Since a straight line is the shortest distance between two points there is no need for nerve transplantation. End to end suture is employed.

Based on that observation, I felt a little tired of having all these children brought back to me every six months, and I determined to do something about it. It occurred to me that if I were to perform my simple mastoidectomies endaurally it should make it impossible for this to happen, because they would not have the postauricular scar to become inflamed secondary to a recurrent middle ear infection. A normal healthy postauricular skin and periosteum would offer more resistance to a postoperatively occurring middle ear infection than the tympanic membrane, and this membrane would instead become inflamed, bulge and rupture as it did before the original mastoidectomy was performed.

LEMPERT ENDAURAL MASTOIDECTOMY.

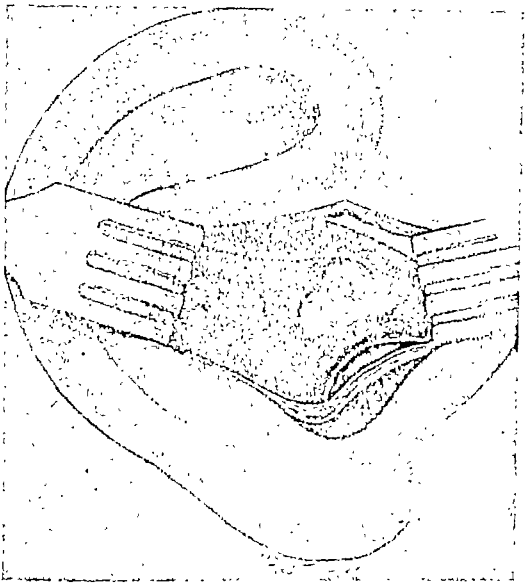

Fig. 30. Completely exenterated mastoid process is seen.

TECHNIQUE.

I have been performing my mastoidectomies for acute suppurative mastoidtis through the endaural approach for more than 20 years. The outline of the anterior cartilaginous margin of the concha underneath the cutaneous cover is brought into prominence by placing the index and middle fingers of the left hand tightly against the face of the concha and pulling and stretching the concha backward against and over the outer mastoid cortex. With the point of the knife directed backward towards the outer mastoid cortex, instead of inward and parallel with the course of the posterior canal wall, the

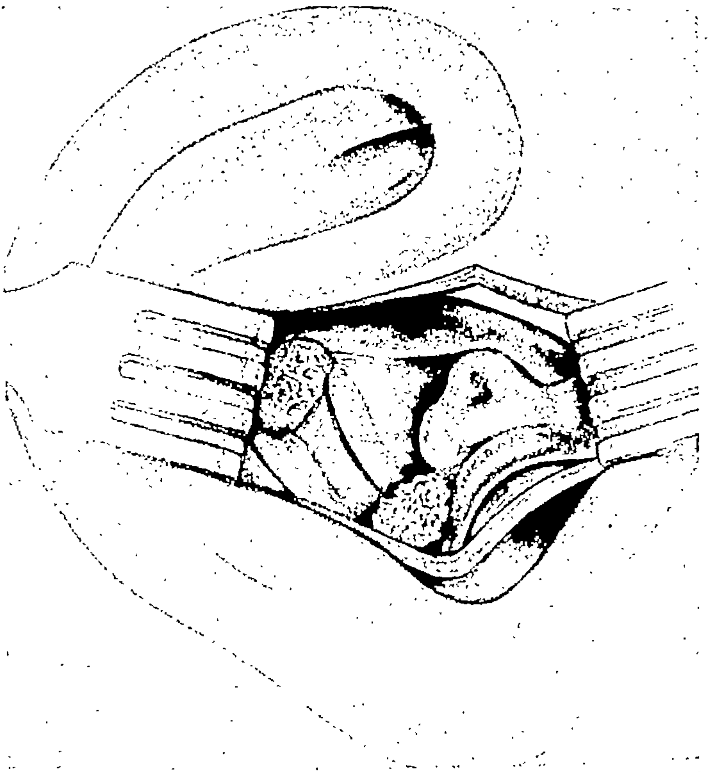

Fig. 31. The lateral sinus is tamponaded at its entrance to and exit from the mastoid process. Lateral sinus wall is incised and thrombus removed from its lumen.

antauricular endaural incision through skin and periosteum is carried immediately adjacent to the entire anterior margin of the concha and upward along the anterior aspect of the helix up to the apex of the suprameatal membranous triangle. When this endaural incision is made correctly an exposure of the mastoid process is obtainable, after subperiosteal elevation of the skin and periosteum from the outer cortex and insertion of the retractors, which is more adequate for surgical intervention than the exposure obtainable as a result of a postauricular incision.

Through this endaural approach I have always without difficulty surgically treated the intracranial complications such

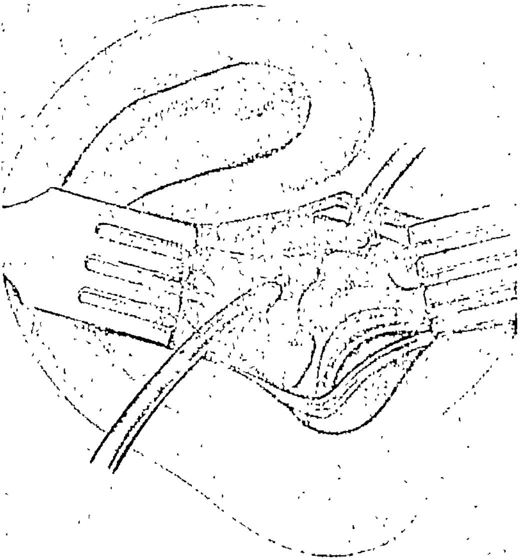

Fig. 32. Middle and posterior fossa abscess is drained with rubber tubing.

as sinus thrombosis, temporosphenoidal abscess, and cerebellar abscess when encountered, and without supplementing the postauricular incision. Today, when the fenestration operation is being performed endaurally by otologists everywhere, the possibility and desirability of doing all temporal bone surgery through the endaural approach is no longer questioned (see Figs. 30-32).

ENDAURAL MASTOIDOATTICOTOMY (MODIFIED RADICAL).

One of the frequently encountered clinical otologic entities requiring surgical attention is the one caused by a chronically persisting and progressive suppurative lesion within the mastoid antrum and epitympanum without having involved the tympanum and hypotympanum. Though the clinical picture presented by this type of otologic lesion and the physical findings are too frequently much more innocent looking than that of a chronic suppurative lesion within the tympanum and hypotympanum, the danger of intracranial complications is greater in the former than in the latter.

As a rule a patient suffering from a chronic suppurative epitympanic lesion gives a history of periodic intermittent purulent middle ear discharge for a number of years which resisted all medical therapy. Physical examination during the period of active middle ear discharge will reveal a fairly normal looking tympanic membrane plus a small perforation in the region of Shrapnell's membrane. If such a patient happens to present himself for examination and advice during one of his dry periods, the perforation in Shrapnell's membrane can be easily overlooked since it is likely to be covered by a small dry crust which gives the appearance of that of cerumen; however, the careful removal of this crust with the aid of a magnifying loop will soon uncover and reveal a small perforation in Shrapnell's membrane and a drop of pus escaping through the perforation into the external auditory canal will soon become apparent. With the escape of this drop of pus tiny shreds of cholesteatoma will become visible within the confines of the epitympanum. In the well advanced case of epitympanic suppuration the patient may give a history of one or more recent attacks of vertigo. In the presence of such a history the slightest pressure exerted upon the region of the perforation with a cotton applicator will evoke severe nystagmus and vertigo. Fistulas of the vestibular labyrinth which rarely accompany chronic suppurative lesions of the tympanum and hypotympanum in the presence of a large central perforation of the tympanic membrane commonly occur in chronic suppurative lesions of the epitympanum with small perforations in Shrapnell's membrane. This is due to the prolonged pressure exerted in the region of the external semicircular canal by the gradually increasing cholesteatomatous mass which surrounds the incudomalleolar joint. The hearing loss for airborne sound accompanying epitympanic lesions is as a rule insignificant and rarely descends to a level lower than 30 db for the three speech frequencies, whereas the hearing loss accompanying chronic suppurative lesions involving the tympanum and epitympanum is as a rule much greater. That surgical eradication of the pathologic process is the only available means of permanently curing this type of chronic suppurative otitis media was well recognized by otologists for very many years; however, methods employed for eradicating the pathology in this type of case were either too radical or inadequate. Even the most adequate and conservative methods employed years ago were in many instances inconsiderate of the future welfare of the ear involved. Many otologists were too radical in their surgical intervention, because they would invariably perform a radical mastoidectomy in such cases with no consideration for the further loss of hearing which must result from such surgery.

Other otologists in their desire to conserve the still existing fairly good hearing would perform what they called a more extensive mastoidectomy and widened the communication between the aditus and antrum, by removing the zygomatic cells. Such surgical intervention was inadequate because it did not provide the necessary accessibility for the removal of the cholesteatomatous mass surrounding the incudomalleolar joint which wound itself around it like cotton threads around a spool. Some well trained and experienced otologists would perform a thoroughly adequate mostoidoatticotomy and successfully remove all the pathology, but they also removed the incus. Though the removal of the incus in such cases need not always result in a further loss of hearing, such removal, however, prevents the occasional improvement in hearing acuity which follows the removal of the pathology which partially immobilized the ossicular chain; furthermore, in the presence of a fistula in the vestibular labyrinth, which is not an infrequent finding in this type of chronic suppurative otitis media, the mastoidoatticotomy technique employed in those days did not provide means for sealing and protecting the membranous labyrinth in the fistula region against degenerating with subsequent total loss of hearing or from becoming infected in the distant future, with subsequent intracranial extension of the infection. Though the immediate welfare of the ear involved was provided for, its distant future welfare was not protected.

Based upon the above mentioned observations made with the surgical treatment of the chronic suppurative lesions within the epitympanum, I developed and employed for the last 14 years the following endaural mastoidoatticotomy technique:

TECHNIQUE.

In perfecting the mastoidoatticotomy technique I considered it essential that the following three objectives be reached as a result of this surgery:

1. The removal of the pathologic process from the epitympanum must be accomplished without disturbing the ossicular chain or injuring the tympanic membrane, so that the preoperative hearing acuity could be conserved. Postoperative further loss in hearing must be avoided.

2. The postoperative mastoid wound must be permanently segregated and excommunicated from the middle ear to prevent the spread of any subsequent middle ear infection to it and its involvement by it.

3. In the presence of a labyrinthine fistula, which is a common finding in such cases, a means of permanently sealing such a fistula to assure the survival of the membranous labyrinth must be provided so that the still existing hearing function may be conserved.

After performing a complete endaural mastoidectomy and exenterating all the pathologic processes encountered within the mastoid process, the aditus is widened, and the mastoid aspect of the posterior, superoposterior and superior bony walls of the external auditory canal is skeletonized. This results in the exposure to view of the incudomalleolar joint and the pathologic processes surrounding it within the epitympanum. The posterior, superoposterior and superior bony walls of the external auditory canal are removed from the skin and periosteum adherent to it, without either lacerating the cutaneous lining of the canal or severing it from its attachment to the tympanic membrane. The pathologic processes encountered within the epitympanum are carefully removed without disturbing the ossicular chain or injuring the tympanic membrane. A viable cutaneous pedicle flap is plastically fashioned from the skin and periosteum from which the bony walls of the external auditory canal have been previously removed, taking care not to sever the cutaneous flap from its attachment to the tympanic membrane. The tym-

LEMPERT ENDAURAL MASTOIDOATTICOTOMY.

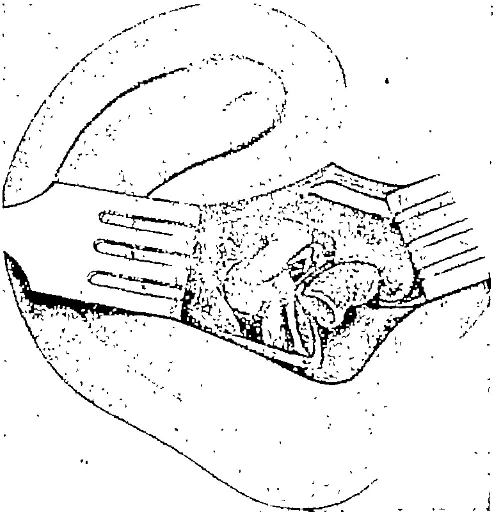

Fig. 33. The pathologic lesion from the epitympanum and mastoid process was removed. The viable tympanomeatal pedicle flap for sealing the tympanic space and isolating and excommunicating the mastoid process from the tympanic space is seen.

panomeatal flap thus created is then carefully trimmed and so placed in the mastoid wound that it will seal off and isolate the epitympanum, the tympanum and hypotympanum from communicating with the mastoid process and will also seal a labyrinthine fistula when present (see Fig. 33).

CONCLUSION.

The viable tympanomeatal pedicle flap which I have created as part of the endaural mastoidoatticotomy technique for the surgical treatment of chronic epitympanic suppuration, suggested and made possible my subsequent creation and development for the first time in the history of otology the now universally employed, practical one-stage fenestration operation for the restoration of practical hearing in clinical otosclerosis.

SUPRAHYOID NECK INFECTIONS.[*][†][‡]

COLBY HALL, M.D.,
Los Angeles, Calif.

Suprahyoid infections differ from other neck infections in certain clinical features. It seems, however, that all infections of the neck share in the confusion of fascial spaces and in the confusion of terminology.

It must be emphasized that the fascial spaces are clinical as well as anatomic. They have become clinical entities because *disease* has invaded them so frequently and has produced such characteristic signs and symptoms. It has been *disease*, rather than regional anatomy, which has given these areas their clinical importance. For example, the peritonsillar space would have attracted little attention had not *disease* dissected between the tonsillar capsule and the pharyngeal aponeurosis. Likewise, the pharyngomaxillary space would have received scant publicity had not pharyngeal *disease* extended laterally beyond the superior constrictor muscle.

With anatomy remaining essentially the same, terminology is important in allowing a common ground for discussion. It becomes necessary, therefore, to state in advance of a clinical report the anatomic area involved and the terminology to be used.

There are several types of suprahyoid infections; however, the author, for this discussion, has chosen the following three: pharyngomaxillary, masticator and Ludwig's.

A brief discussion for the purpose of anatomic orientation and terminology will be followed by a clinical report of cases which are characteristic of infection in each area.

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[*] Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 25, 1950.

[†] From the Department of Otolaryngology, School of Medicine, University of Southern California.

[‡] Original drawings by Mr. Ted Bloodhart, Los Angeles, Calif.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

PHARYNGOMAXILLARY.

When tonsillar or pharyngeal disease extends laterally through the superior constrictor muscle it enters the pharyngomaxillary fossa which was described by Mosher[1] in 1929 (see Figs. 1 and 2). A study of the diagrammatic sections reveals the anatomic basis for the signs and symptoms which may result from disease in this area. The tonsil, if present, and the lateral pharyngeal wall are pushed toward the midline. Pressure laterally against the internal pterygoid muscle results in a certain degree of trismus, while external swelling over the parotid is not infrequent. Should the process reach the inferior limit of the space, at the level of the hyoid bone, induration, swelling and tenderness below the angle of the mandible will result. Most important, however, is the intimate relationship to the contents of the carotid sheath. In addition to a periphlebitis, or perijugular abscess, two other very seri-

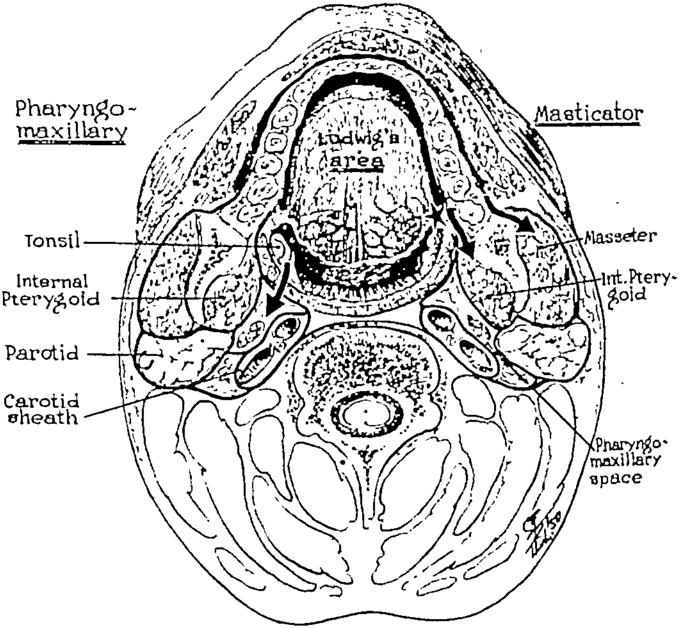


Fig. 1. Diagrammatic Cross Section.
Pharyngomaxillary area and relationships shown on left. Arrow indicates course of disease through the superior constrictor muscle.
Masticator space is shown on right with arrows indicating course of dento-alveolar disease.
Star indicates area of spontaneous drainage in masticator abscess.

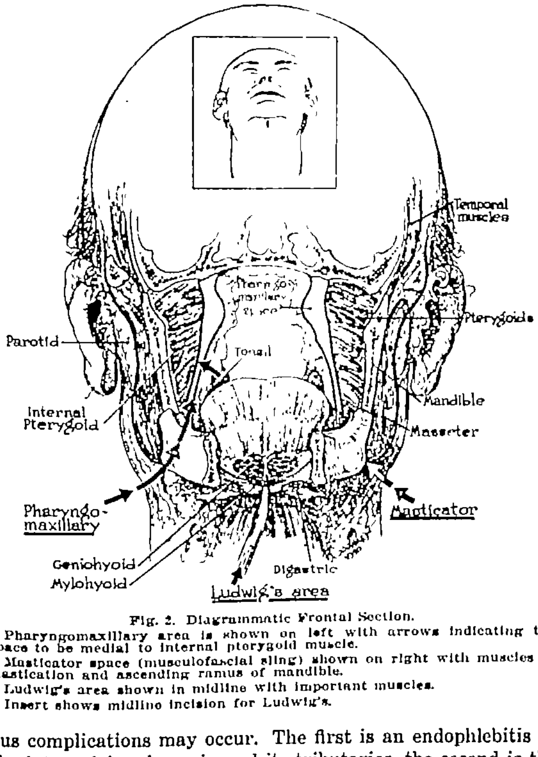


Fig. 2. Diagrammatic Frontal Section.

Pharyngomaxillary area is shown on left with arrows indicating the space to be medial to internal pterygoid muscle.

Masticator space (musculofascial sling) shown on right with muscles of mastication and ascending ramus of mandible.

Ludwig's area shown in midline with important muscles.

Insert shows midline incision for Ludwig's.

ous complications may occur. The first is an endophlebitis of the internal jugular vein and its tributaries, the second is the dreaded erosion which most commonly involves the internal carotid artery.

MASTICATOR.

Dentoalveolar and subperiosteal infections of the mandible have so frequently extended to involve the masseter and internal pterygoid muscles that this area has also become an entity. It is known as the masticator space and was described by Coller and Yglesias[2] in 1934 (see Figs. 1 and 2). A review of the diagrammatic sections reveals this space to be the immediate lateral neighbor of the pharyngomaxillary space; therefore, the signs of disease in this area closely resemble those of pharyngomaxillary involvement. On frontal section (see Fig. 2) this area appears as a musculofascial sling containing the muscles of mastication and the ascending ramus of the mandible. Posterior extension of disease from the molar teeth, most commonly the third molar, may be either along the medial or along the lateral aspect of the mandible. Immediately upon reaching the masseter or pterygoid muscles, definite trismus occurs. The predominance of internal or external swelling will depend upon whether the pathology is most marked along the lingual or along the buccal margin of the mandible. Infection in the masticator space often produces, in addition to extreme trismus, induration involving the posterior sublingual tissues and the lateral pharyngeal wall.

LUDWIG'S.

The term Ludwig's angina is so permanently implanted in our medical literature that it may appear academic to be controversial regarding the term; however, due to its varied and often promiscuous usage, it becomes imperative that one state what he considers to be Ludwig's and what anatomic area is involved. The author agrees with the very concise and definite description of Ashurst.[3] He stated, "It is an acute inflammatory process involving the cellular tissues of the floor of the mouth and the submaxillary region of one or both sides of the neck. It is the simultaneous involvement of the submaxillary and the sublingual tissues in a confluent septic cellulitis that warrants the condition being recognized as a distinct clinical entity." He further stated that it may begin in either the sublingual or the submaxillary area, but until it involves both it does not constitute Ludwig's. The term "submaxillary region" should be emphasized, for this is not primarily a

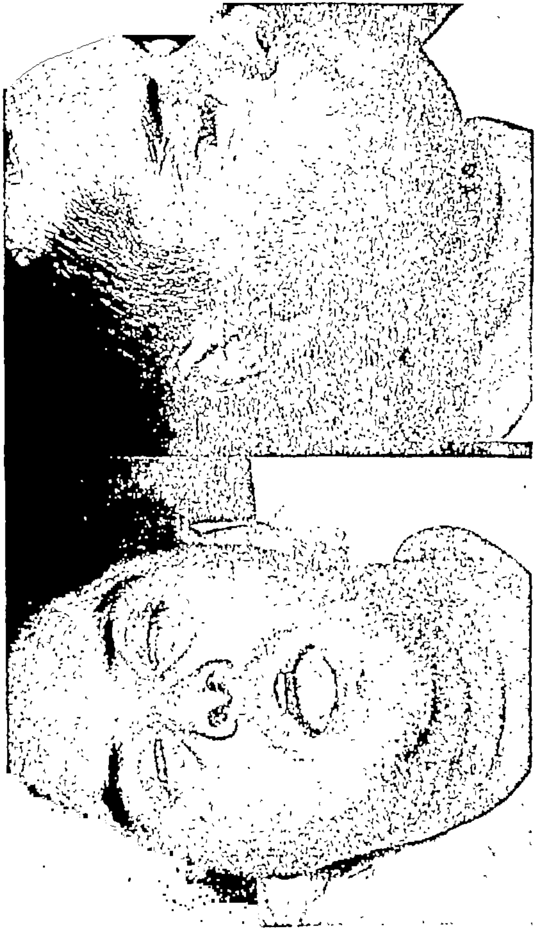


Fig. 3. Pharyngomaxillary Abscess. Upon admission to hospital.

submaxillary gland infection. Infections of this type are usually of dental origin.

The thin mylohyoid, because of its wide bony insertions, presents a formidable muscular diaphragm (see Fig. 2). Above it lie the thick geniohyoids and the muscular tissues of the tongue itself. A study of this musculofascial area with its unyielding attachments to the mandible and to the hyoid bone readily reveals how a spreading cellulitis may produce alarming symptoms due to respiratory embarrassment, tissue necrosis, sepsis and possibly to carotid sinus involvement.

PHARYNGOMAXILLARY ABSCESS.

The first case to be reported is that of a pharyngomaxillary abscess. This white male, age 35, complained of a mild soreness in the right side of the throat on Aug. 24, 1949. The following day he was symptom-free and remained so until Aug. 28. Upon this date, four days following the mild soreness, there was extreme pain in the right throat, there was rapidly increasing swelling about the parotid and the mandibular angle, and there was intense malaise. He was admitted to the hospital on this same day, where examination revealed the following important signs: temperature 105°, leucocytosis 26,000, moderate trismus, generalized swelling over the right side of the face and below the mandible (see Fig. 3) and marked medial displacement of the right tonsil and pharyngeal wall. There was no acute pharyngeal inflammation. The right hypopharyngeal wall was displaced medially to such an extent as to produce respiratory embarrassment. On Aug. 29, five days after the initial symptom, and one day following the marked symptoms, a preliminary tracheotomy was performed. With an adequate and safe airway established, pentothal anesthesia was administered for the incision and drainage.

A transverse incision was made at the level of the hyoid bone beneath the angle of the jaw. After cutting through the edematous subcutaneous tissue the cervical fascia was exposed and found to be very tense. When this fascia was incised no pus was encountered, *but* upon inserting a curved forceps upward medial to the mandible, foul pus escaped under pressure (see Fig. 4). A Penrose drain was then inserted to the

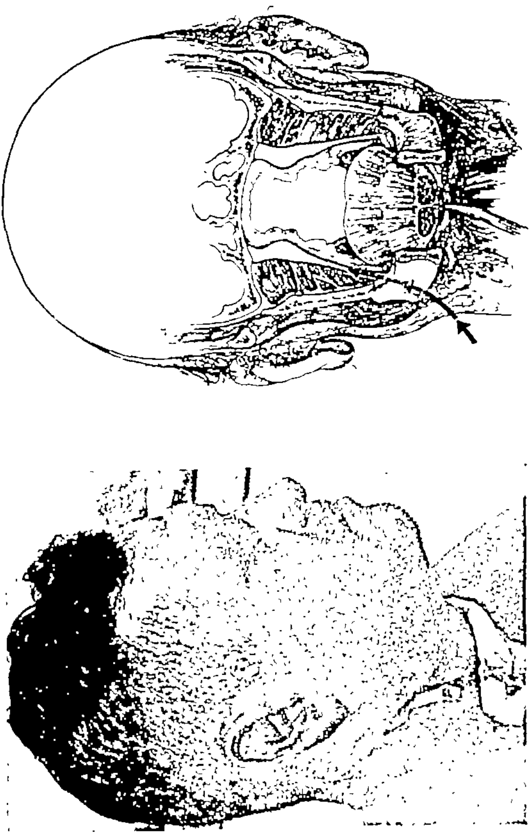


Fig. 4. Pharyngomaxillary Abscess. Arrows indicate location of pus and course of Penrose drain.

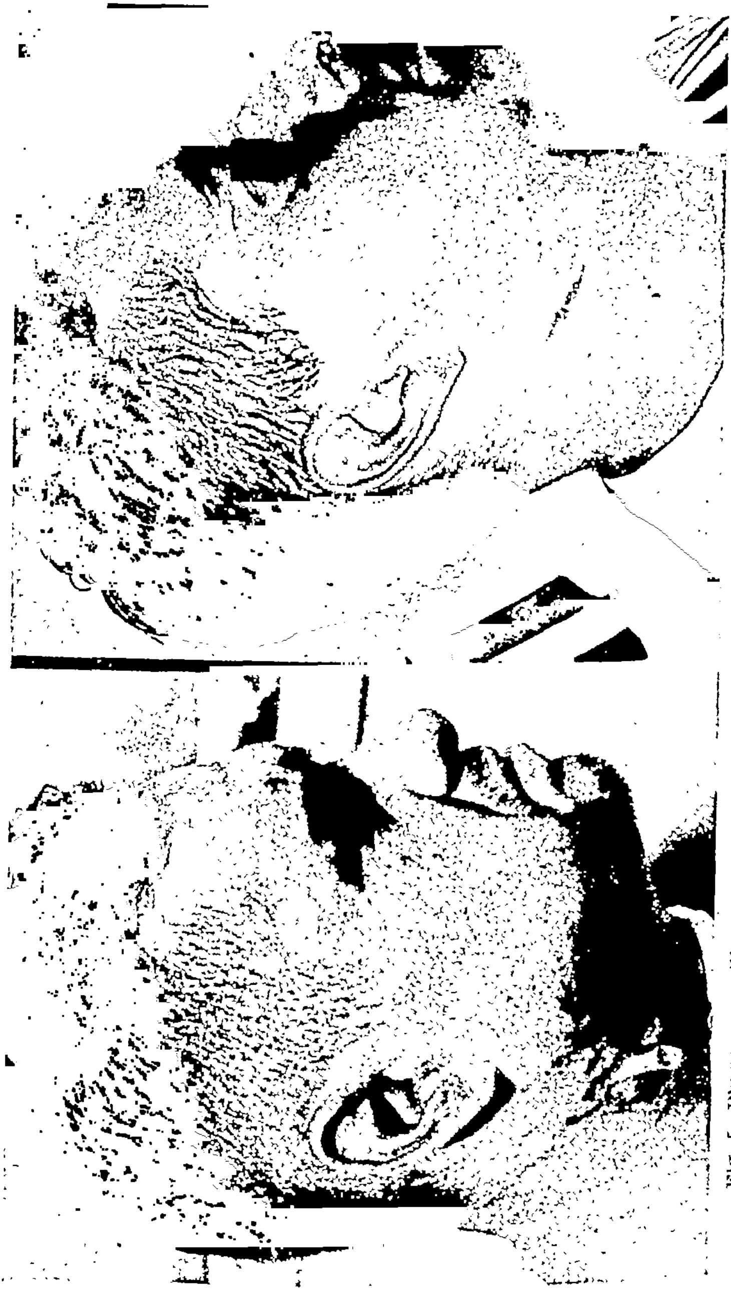


Fig. 5. Pharyngomaxillary Abscess. Left, 24 hours postoperatively. Right, three weeks postoperatively.

depth of the wound. Intensive antibiotic therapy was continued and the patient made a rapid and uneventful recovery (see Fig. 5).

The important points to be emphasized are: pharyngeal history, pharyngeal wall displacement without acute pharyngeal inflammation, rapidly increasing and severe clinical picture, only moderate trismus, easy depression of the tongue on both sides posteriorly, no fluctuation, and the finding of pus medial to the jaw and to the internal pterygoid muscle.

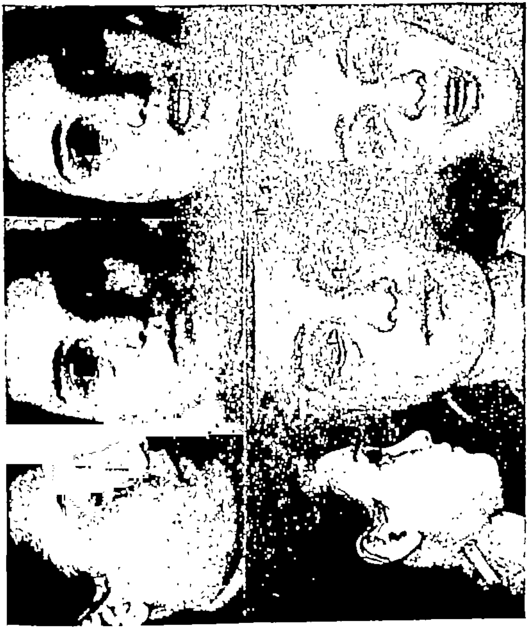


Fig. 6. Masticator Space Infection. Upper, prior to external drainage. Right photo shows patient attempting to open her mouth. Lower, 48 hours following external drainage.

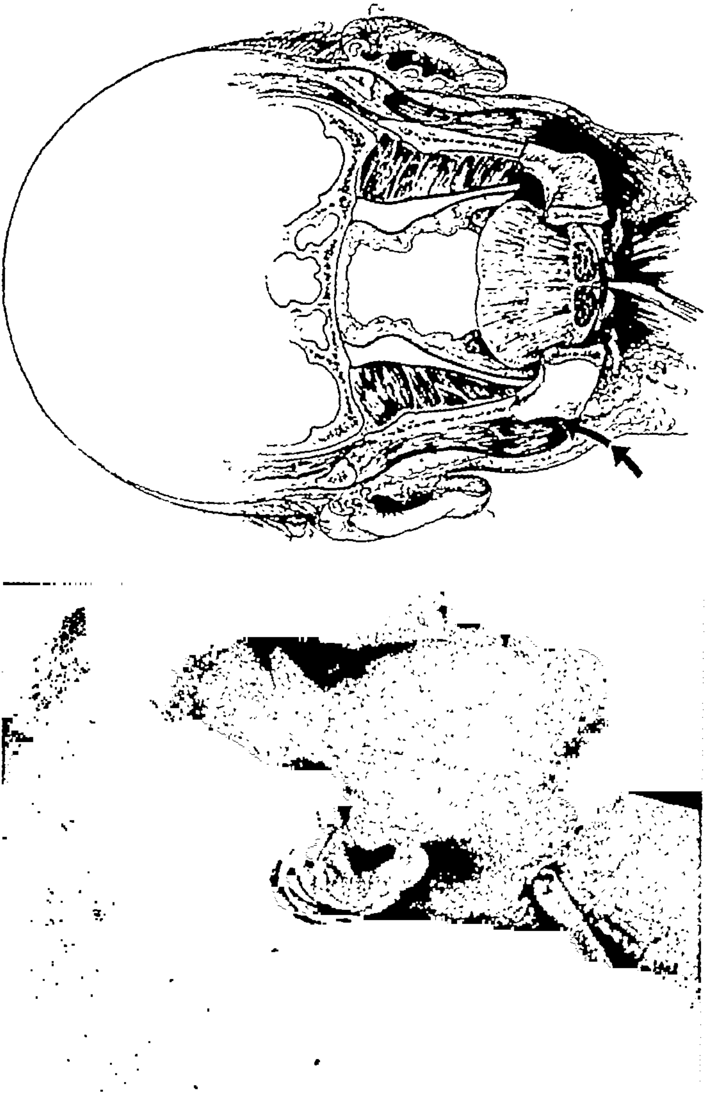


Fig. 7. Masticator Space Infection. Arrows indicate location of pus and position of Penrose drain.

MASTICATOR SPACE INFECTION.

The second patient presented disease in the masticator space.

This girl, age 20, was first seen on Jan. 29, 1949, and presented a painful swelling over the right jaw associated with trismus of moderate degree, and temperature of 101.5°. Two weeks previously she had complained of a right-sided toothache which subsided following spontaneous drainage into her mouth. On Jan. 31, two days after admission, an intraoral incision was made along the mandible opposite the right lower third molar. Pus was recovered, and she was discharged on Feb. 2.

Two week later, Feb. 15, she was readmitted with marked trismus, difficult depression of the tongue on the affected side posteriorly, and tender swelling over the jaw externally (see Fig. 6). There was no fluctuation. External incision was made below the angle of the jaw and when this wound was explored *to the bone*, pus was recovered (see Fig. 7). Dental radiographs revealed an apical abscess of the right lower third molar. On Feb. 28 the offending third molar was extracted. One month following the surgical drainage the trismus and swelling had entirely subsided. It should be remembered that subsidence of trismus in these masticator cases is usually rather slow.

The important points to be emphasized are: dental history, extreme trismus, the posterior portion of the tongue on the affected side was impossible to depress, no fluctuation, the patient was not acutely ill as compared to pharyngomaxillary abscess, and the abscess was *on the bone*.

Intraoral incisions along the mandible are sometimes sufficient, but when they prove otherwise, as in this case, external drainage must be performed. In masticator abscess the pus is on the bone rather than medial to the pterygoid as in pharyngomaxillary abscess (see Fig. 2).

A large percentage of masticator space infections drain spontaneously within 10 days of the swelling, as reported by Hall and Morris.[4] When this occurs pus can be seen escaping from an area between the base of the tongue and the mandible (see Fig. 1).

LUDWIG'S.

The third patient, age 32, presented what we considered to be Ludwig's infection.

Her right lower third molar was extracted on May 16, 1949. This was followed by marked external swelling within 48 hours. A dentist performed a submental incision at this time and penicillin was administered. She entered the hospital on May 23. She was acutely ill. The temperature was 104°, and there was intense board-like swelling in the entire submaxillary region (see Fig. 8). The tongue was extremely swollen and pushed upward against the palate (see Fig. 9), and the oral floor was board-like. There was no fluctuation. There was dyspnea, dysphagia, dysphonia and drooling. The incision was immediately extended upward to the floor of the mouth. A small curved forceps was used to separate the tissues on either side of the wound. Only a scant amount of thin secretion was recovered, although some days later there was a moderate purulent discharge. Intense antibiotic therapy was continued. The symptoms rapidly diminished, and the patient was discharged one week later (see Fig. 8).

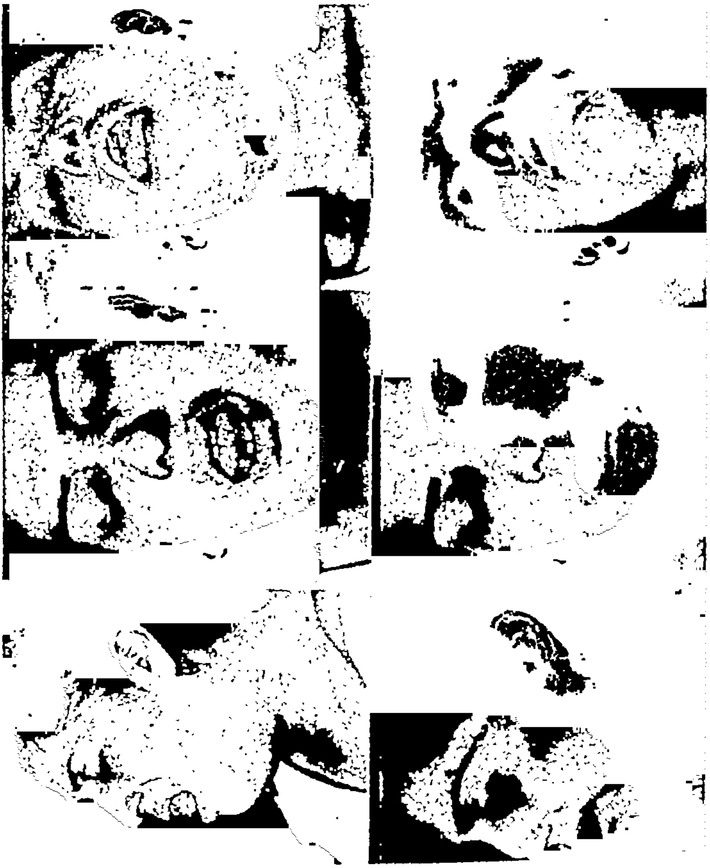


Fig. 8. Ludwig's Infection. Upper, upon admission to hospital. Coexistence of disease in sublingual and submaxillary regions. Lower, one week later upon discharge.

The photographs (see Figs. 8 and 9) demonstrating the coexistence of disease in the sublingual and submaxillary regions appear to be typical of Ludwig's infection as we know it. No doubt there have been more fulminating cases, but the anatomy, the etiology, the signs and the treatment remain the same.

We prefer an incision similar to that described by Dr. August Beck[5] in his complete work on Deep Neck Infections.

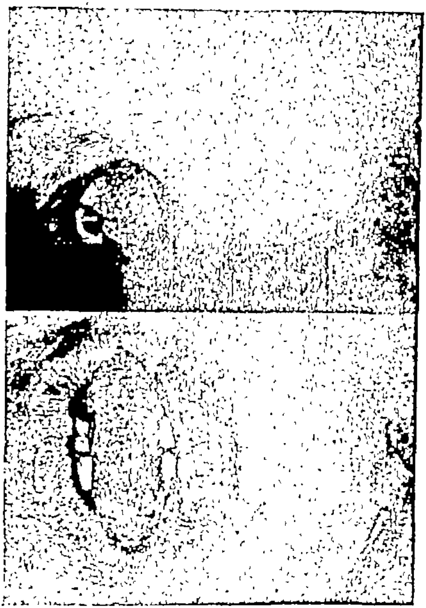
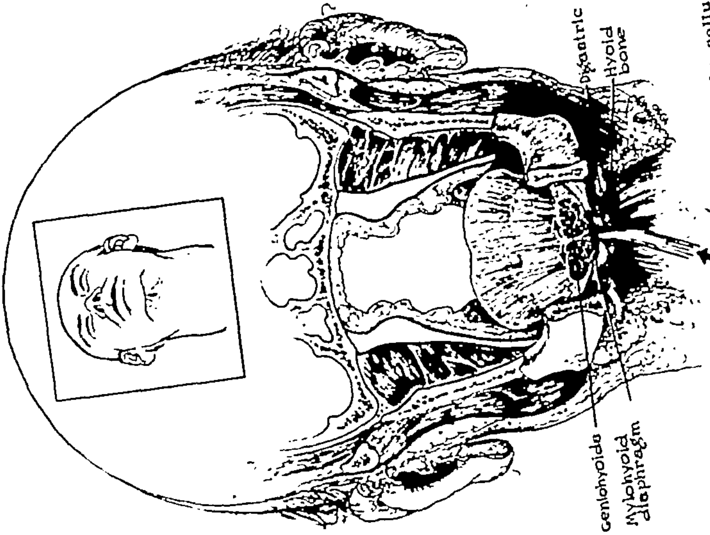
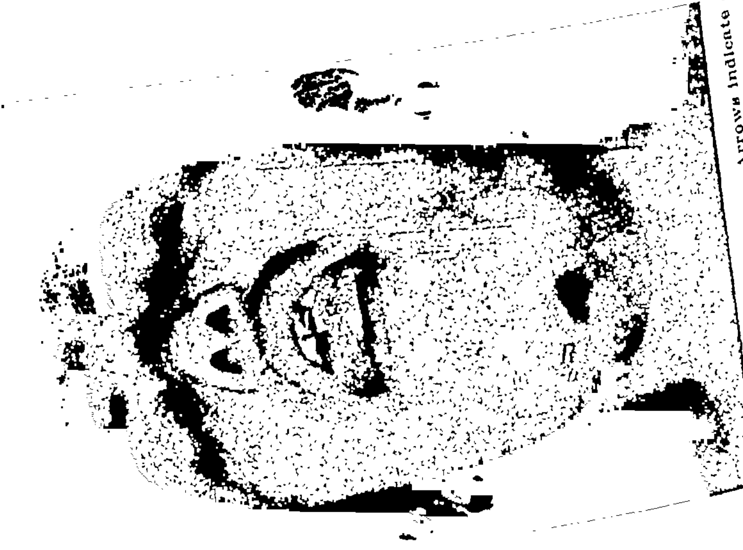


Fig. 9. Ludwig's Infection. Tongue fills the mouth

A midline incision is made between the mandible and the hyoid bone. It is then carried upward between the digastrics and through the mylohyoid. The incision is extended upward between the geniohyoids with separation of the inflammatory tissues on either side of the midline with curved forceps for the purpose of decompression and drainage (see Fig. 10). This procedure has been effective in a large number of cases with prompt relief of the respiratory and other symptoms. There is no objection to performing a preliminary tracheotomy, after which a general anesthetic may be employed. Early recognition of Ludwig's is imperative, for there are many who feel that early decompression is essential as a preventive of tissue necrosis.

The cases reported demonstrate three important points which may be applied to all deep infections of the neck:





Arrows indicate course of forceps in entering and decompressing the cellulitic

1. Antibiotic and chemotherapy should be used from the onset, but these agents are no substitute for drainage. Should serious symptoms supervene, surgical drainage or decompression must be performed.
2. Fluctuation is important when present, but should never be awaited.
3. An adequate airway is essential. If a safe airway is present, or can be established, a general anesthetic may be administered.

I am quite aware that there are all stages, degrees and variations of the types of infections reported above. Whether incipient cases are recognized as such is of little importance as long as the patients recover.

When the anatomy of the pharyngomaxillary, masticator and Ludwig's areas is applied to the signs, symptoms and treatment of the cases reported, it appears that each is characteristic of infection in its respective area.

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THE MANAGEMENT OF NASOPHARYNGEAL FIBROMAS.*

FREDERICK A. FIGI, M.D.,† and ROBERT E. DAVIS, M.D.,‡
Rochester, Minn.

In previous reports on nasopharyngeal fibromas the successive changes made in the methods of treating these tumors and the greater safety and effectiveness of these measures have been discussed. One of these communications presented the experience of our colleagues, and one of us (Figi) in dealing with 63 cases of this type encountered at the Mayo Clinic from Jan. 1, 1910, through Dec. 31, 1939. The outstanding observation noted in this study was that in 73 per cent of the cases the tumors completely disappeared before the patients attained the age of 20 years, which is approximately the time when fibromas of this type may be expected to show initial evidence of spontaneous regression. From Jan. 1, 1940, through Dec. 31, 1949, 51 additional cases of nasopharyngeal fibromas came under our observation, and these form the basis for the present study; however, the remarks and conclusions which follow reflect our overall experience with the total of 114 cases.

Nasopharyngeal fibromas, also commonly known as "juvenile basal fibromas of the nasopharynx," "fibroid tumors of the base of the skull," "fibroangiomas of the nasopharynx" and "juvenile nasopharyngeal fibromas," are uncommon in spite of this rather large group of cases. Shaheen,[1] in 1930, reported 58 cases under the title of "nasopharyngeal fibromas"; however, in view of the extremes in age of some of the patients in his series, which ranged from two to 70 years, it

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif, May 27, 1950.

†Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic, Rochester, Minn.

‡Fellow in Surgery, Mayo Foundation, Rochester, Minn.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

seems obvious that a number of these lesions were not of this nature. Recently Härkönen and Malmio[2] reported 31 such cases from Stockholm, and Martin, Ehrlich and Abels[3] presented a study of 29 cases seen on their service at the Memorial Hospital in New York. With these exceptions, practically every series of fibromas of the nasopharynx recorded in the literature has comprised fewer than 10 cases; accordingly, the development of present-day therapy has necessarily come from the accumulated experience of different observers.

The onset of these fibromas is definitely limited to the period of adolescence, and this is one of their most striking features. Fibrous tumors presenting somewhat the same appearance, but which usually do not behave like true juvenile nasopharyngeal fibromas, are occasionally seen in the nasopharynx, nasal fossae and accessory sinuses of patients who have not yet reached the age of puberty, and also in young adults, and in persons of advanced years. Confusion has resulted in a number of instances, for such lesions have at times been reported in the literature as juvenile basal fibromas. Since they are quite innocuous in comparison with the highly vascular, locally invasive lesions under consideration, it is essential that they be differentiated.

When the patient who has a juvenile nasopharyngeal fibroma attains sexual maturity, or roughly at about the age of 20 to 25 years, the tumor generally becomes less active than before and shows a varying degree of natural regression; however, as stated in a previous communication, we have never observed the complete spontaneous disappearance of these fibromas that is so often referred to in the literature. At the same time, it should be noted that because of the locally destructive character of such tumors, and their proximity to a number of vital structures, we have not felt justified in marking time and idly observing them to see whether complete recession would occur; moreover, most of our patients who had nasopharyngeal fibromas have lived at considerable distances from the clinic, and their finances have been too limited to permit them to return for observation more frequently than the situation demanded. Rarely these growths do persist and continue to progress after the patient reaches sexual maturity. Included in the present series is a man who came under our care at the age of 28 years with an extensive basal fibroma of the nasopharynx and right nasal fossa that had first produced symptoms at the age of 23 years and which had recurred following repeated previous removal.

The predominance of these tumors in males and their association with adolescence have naturally given rise to hypotheses concerning a probably etiologic relationship with a disturbance of sexual development. Actually, however, little more is definitely known about their cause than that of neoplasms of other types.

Juvenile basal fibromas generally grow slowly through the period of sexual development, although there may be intervals of markedly accelerated activity due to an acute inflammatory process, trauma, or for no obvious reason. While these neoplasms are perfectly benign histologically, and never metastasize, they may be highly destructive locally and often produce severe debility. Because of their proneness to cause pressure absorption of bone and to send out processes which invade and enlarge normal ostia and bony fissures, any one or all of the nasal accessory sinuses, the retromaxillary fossa, the orbit, and even the cranial cavity may be invaded.

As has been repeatedly emphasized by writers on this subject, the tumors under consideration occur predominantly in males; in fact, the present group of 51 patients consists entirely of males, and Martin[3] and his colleagues, from their study of 29 cases, voiced the conviction that the neoplasm never occurs in females; however, the personal series of 63 cases reported 10 years ago included five females, and a recent critical review of these convinces us that they definitely belong to this category. Other authors also have noted the infrequent occurrence of these tumors in females. Thomson and Negus[4] stated that they are rare in girls, and one of the illustrations in their textbook is a photograph of a young Negress with a tumor of this type protruding from the nostril and producing the characteristic "frog-face" deformity often associated with advanced neoplastic involvement of the nasal fossae.

Among the 51 patients reported here, the age of onset, with one exception, ranged from nine to 17 years, averaging 13.6 years; the age at the time of examination ranged from 10 to 20 years, averaging 15.1 years; and the age at which the tumor was arrested ranged from 10 to 21 years, averaging 16.9 years (see Table 1). In the one exception referred to previously, symptoms had first been noted about the age of 23 years, and the tumor was continuing to progress actively at the time of our examination at the age of 28 years. There was not the slightest doubt in our minds concerning this tumor's being a juvenile basal fibroma, and the experienced laryngologist who had treated the patient previously was of the same opinion. Depending upon the site of origin and the rate of growth, it seems likely that the lesion may remain symptomless and entirely silent for a period ranging from a few months to several years. In the present series the patients had complained of symptoms, or signs had been observed by others, for from one month to several years, averaging 18.2 months prior to our examination. An interesting observation in this connection is that reported by Martin and his associates of an asymptomatic nasopharyngeal fibroma discovered in a boy aged 15 years during routine physical examination. The tumor was observed by them for two and one-half years, and they noted no appreciable change in its size. Of interest here also is a symptomless lymphosarcoma of the nasopharynx

TABLE 1.
NASOPHARYNGEAL FIBROMAS. AGE OF PATIENTS.*

Age	*Range, Years*	*Average Years*
At time of onset	9-17	13.6
When first seen at clinic	10-20	15.1
When tumor arrested	10-21	16.9

*Exception: One patient not included, whose symptoms began at age 23 years.

seen at the Mayo Clinic some years ago in a woman 61 years of age.

The clinical findings in cases of nasopharyngeal fibroma are quite uniform. Examination with a nasopharyngeal mirror shows a hard, rounded or slightly nodular, deep red, purplish or grayish tumor filling a varying portion of the nasopharynx and often extending into one or both nasal fossae as well. A striking feature is the marked vascularity of the overlying mucous membrane, numerous prominent tortuous vessels generally being evident. Ulceration of the mucous membrane is unusual unless there has been recent trauma. The attachment is usually broad, sessile and firm, and the mass generally is quite immobile. Rarely the tumor has a definite pedicle, and its lower portion infrequently is visible below the palatal border. Secondary attachments may be present if previous attempts at removal have been carried out. Anterior rhinoscopy usually shows a varying portion of one nasal fossa filled with neoplastic tissue, which at times crowds the septum well over. The mass may be present in the nasal vestibule or even protrude from the nostril (see Fig. 1). There may be broad-

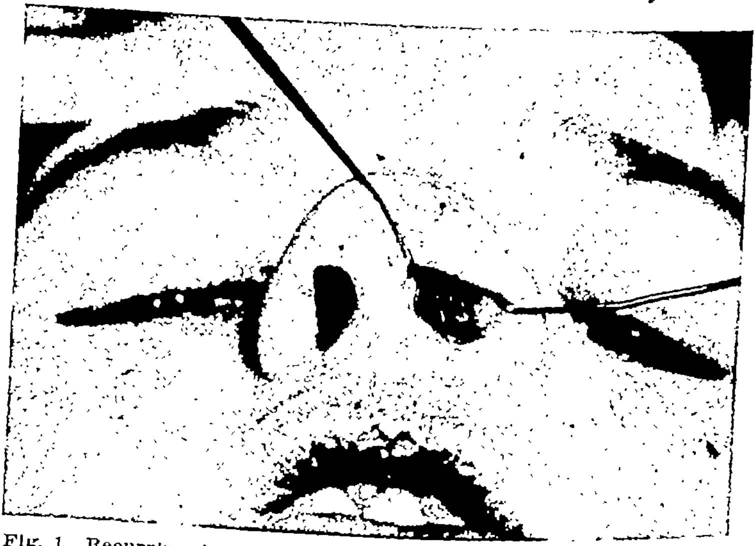

Fig. 1. Recurring juvenile fibroma of left nasal fossa and nasopharynx presenting in the left nostril.

ening of the nasal bridge and root, bulging of the cheek, or elevation and proptosis of the eye, due respectively to involvement of the upper part of the nasal fossa, the ethmoid labyrinth, the antrum, the retromaxillary fossa or the orbit.

In a previous report on this subject, it was stated that fibromas of the nasopharynx provide one of the few exceptions to the generalization that biopsy should be made in all cases of neoplasm occurring in the upper part of the respiratory tract. Our feeling concerning this is unchanged. An experienced laryngologist who has seen a few nasopharyngeal fibromas can, as a rule, recognize them quite definitely on clinical examination. The gross appearance of the tumor as seen in the nasopharyngeal mirror, or on direct inspection of the nasal fossa, is very characteristic; however, if the lesion is growing actively, is recurring following recent removal, or is acutely inflamed, the clinical picture may be highly suggestive of a malignant neoplasm, and biopsy becomes essential. When any question has existed in this regard, we have routinely performed biopsy in these cases; otherwise it has been avoided. In the present series biopsy was performed in a little more than one-half of the cases. If the diagnosis is obvious from gross inspection, it is advantageous to refrain from removal of tissue for microscopic study, as this procedure often gives rise to profuse bleeding which is difficult to control, either by firm packing or by electrocoagulation. At times performance of a biopsy has resulted in such serious loss of blood that ligation of the external carotid artery and transfusion have been required, and removal of the tumor has been rendered much more difficult. In one of the patients in this series electrocoagulation was rendered impossible by the profuse bleeding induced by biopsy, and ligation of the external carotid artery and transfusion were necessary.

Malignant change in nasopharyngeal fibroma has been reported by some observers, and Thomson and Negus[4] considered this a possibility. Until recently we have been skeptical concerning the likelihood of this occurrence; however, we have recently had under observation a man, 20 years of age, from whose nasopharynx, nasal fossae and sphenoid sinus we removed a recurring vascular fibroma, 4 cm. in diameter, three and one-half years ago. This patient has at no time presented obvious recurrence of his tumor, but for the past two and one-half years has at intervals experienced moderately severe right frontal and parietal headaches. During recent months these had become increasingly severe and right external rectus palsy and dimness of vision developed, which progressed to total blindness of both eyes. Roentgenograms showed what appeared to be malignant destruction anterior to the sella. The neurologic consultants on our staff were confident that the condition was malignant, and considered it inoperable; however, the possibility of an inflammatory process could not be definitely excluded.

The treatment of nasopharyngeal fibromas has undergone considerable change during the past few decades. Primarily therapy was entirely surgical, but experience generally with such measures was a high mortality rate and frequent recurrence necessitating repeated operations. Consequently, observation of the effectiveness of radiation in dealing with these lesions resulted in its almost complete replacement of surgical procedures for a period. More recently the combination of surgical diathermy and radiation, the latter preferably used interstitially, has been found to produce the most satisfactory results. Surgical exposure of the tumor frequently is necessary in order to permit applying these measures with greatest effectiveness.

In all but nine of the 51 patients in this series, the fibromas had either been removed surgically, or treated with radiation, or some surgical diagnostic procedure had been carried out prior to our examination (see Table 2). Removal surgically or with electrocoagulation or both had been carried out in 28 patients on from one to six occasions. Nine had received interstitial radiation from one to four times; 15 had been given Roentgen therapy, either alone or combined with surgical measures or interstitial radiation. One patient had received five courses of Roentgen treatment, and another had been given 80 applications of Roentgen radiation. What was

TABLE 2.

NASOPHARYNGEAL FIBROMAS. PREVIOUS TREATMENT.

Cases in this series		51
Cases without previous treatment		9
Cases with previous treatment*		42
Surgical removal or electrocoagulation or both	28	
Interstitial radiation	9	
Roentgen therapy	15	
Biopsy only	3	
Sclerosing injection	1	

*Several patients had been treated by surgical removal and electrocoagulation, as well as by interstitial radiation.

assumed to be some type of caustic or sclerosing solution had recently been injected directly into the tumor in one case. Another patient who came with a huge mass obstructing both nasal fossae complained of residual diplopia dating from an attack of meningoencephalitis that had followed the last surgical removal of his tumor some months previously. In three cases biopsy only had been carried out. In one of these, in which the procedure had been done a week previously, the patient came with firm gauze packs in the nose and nasopharynx. He had bled so severely that he had severe secondary anemia, and it was necessary to admit him directly to the hospital for a transfusion.

Recent surgical procedures generally increase the difficulty of subsequent operative measures and render them less effective. This is due to the fact that they tend to obscure the limits of the residual tumor, decrease its mobility, and often increase its vascularity by producing multiple secondary attachments. On the other hand, intensive Roentgen therapy at times appears to render subsequent treatment, especially with interstitial radiation, more effective. It has seemed in some instances to facilitate electrocoagulation, probably by reducing the blood supply. At least in a number of cases in this group in which the patients had previously received heavy Roentgen treatment, fewer treatments were required to eradicate the fibroma than were necessary in some of the others.

The surgical approach to nasopharyngeal fibromas has a direct bearing on the possibility of complete removal, and accordingly on the prognosis. It necessarily varies with the extent, activity, vascularity and situation of the growth, the nature, site, and area of its attachment, and the previous treatment. Adequate exposure is essential, but often it is difficult to obtain without mutilation, which rarely is justifiable. Such procedures as splitting the soft palate and temporary resection of the maxillae in order to gain access to the tumor have gone into the discard. The approach may make the

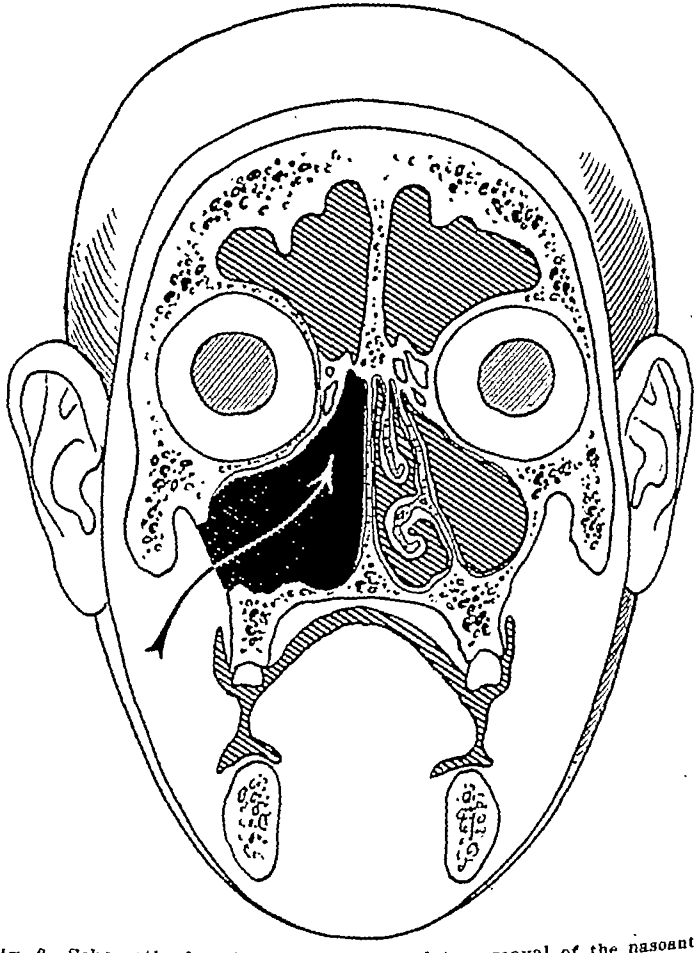

Fig. 2. Schematic drawing showing complete removal of the nasoantral wall during course of transantral approach to tumor of the nasal fossa or nasopharynx.

difference between an extremely hazardous and a reasonably safe procedure. It may permit eradication of the growth in a single operation with a brief convalescence, or necessitate repeated surgical measures or applications of radiation during the course of a number of months or even several years with a much less satisfactory ultimate result.

In the group of nasopharyngeal fibromas reported in 1941, treatment directly to the tumors was carried out entirely through the nostrils and transorally through the nasopharynx. In the present series also a majority of the tumors were dealt with in the same manner; however, during the past three and one-half years a transantral approach has been used to secure access to the neoplasm in a number of these cases, and the procedure has proved highly effective. It entails performing a Caldwell-Luc operation, most of the anterior wall of the antrum being removed, and then taking away a varying portion of the lateral wall of the nose, depending upon the situation of the growth (see Fig. 2). The approach through the nares and nasopharynx usually affords quite satisfactory access when the fibroma is situated in the lower part of the nasal fossa and in the nasopharynx. Better exposure of these regions can be secured transantrally, and this is true also when the tumor is in the superior portion of the nasal fossa, the ethmoid labyrinth, antrum, sphenoid sinus, orbit or retromaxillary fossa; in fact, involvement in many of these situations is inaccessible through the nostrils and nasopharynx and removal by these routes is much less certain and bears infinitely greater hazard than by the transantral approach (see Fig. 3). Many nasopharyngeal fibromas have a broad sessile attachment high on the posterior wall or over the vault of the nasopharynx, and a surprising number are attached on the posterior portion of the lateral wall of the nose, especially high up. All of these regions are readily accessible through a transantral approach, as is also the involvement commonly extending from these regions into the regions mentioned previously; that is, the sphenoid sinus, orbit, and retromaxillary fossa. At times the maxillary sinus is not actually invaded by the tumor, yet the antral cavity is encroached upon and

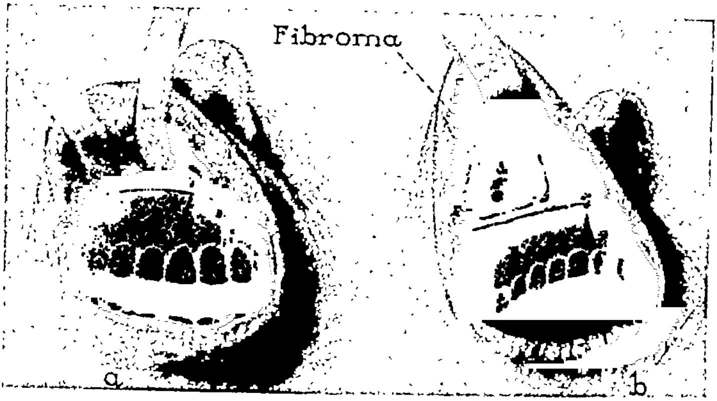

Fig. 3. Transantral approach for removal of juvenile basal fibroma of the nasopharynx and right nasal fossa. The anterior tip of the tumor is presenting in the midportion of the nasal fossa.

reduced in size as much as 75 per cent or more, owing to the mass in the nose or in the pterygomaxillary fossa crowding upon its mesial or posterior walls. Even when this is true, excellent exposure usually can be secured through the antrum.

A vulcanite nasal speculum is used when the diathermy is applied through the nostrils in order to avoid injury to these structures. For coagulation of that portion of the tumor which is situated in the nasopharynx the soft palate is retracted by means of a rubber catheter introduced through either nostril and brought out through the mouth, as described in a previous communication (see Fig. 4). Intravenous pentothal sodium anesthesia is employed in removal of these tumors, and an intratracheal tube is inserted through the mouth to maintain the airway, and for the administration of oxygen. A moist gauze pack placed firmly about this tube holds it to one side out of the line of vision and prevents gravitation of blood into the lower part of the respiratory tract. In some cases, because of the activity, extreme vascularity and extent of the tumor insertion of radium alone through the nostrils and nasopharynx has been carried out, rather than an attempt made at surgical removal. With extension of the growth well laterally in the retromaxillary fossa and bulging of the cheek and soft palate, implantation of radon seeds is at times made directly through the posterior portion of the buccal fold and through the soft palate.

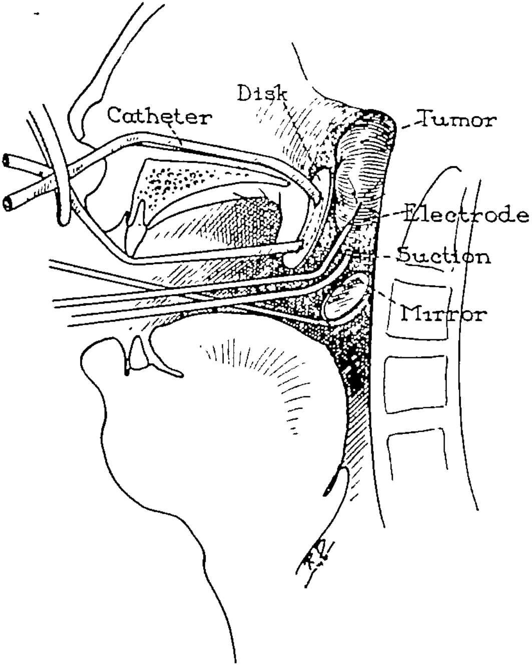

Fig. 4. Method of exposing portion of tumor in nasopharynx. (From Figi, F. A.: Fibromas of the Nasopharynx. Jour. A. M. A., 115:665-672, Aug. 31, 1940.)

Much the same procedure is followed when the tumor is approached transantrally except that the better exposure usually obtained permits more accurate determination of the limits and relationships of the tumor and facilitates its removal. Although many of these tumors are quite firm and present as a discrete, rounded, nodular mass, some of them are quite soft and friable, and the capsule is thin and delicate and tears readily on slight traction. Others present accessory lobules or processes of varying size which become detached easily, rendering clean removal difficult, and often giving rise to recurrence. Providing hemostasis is reasonably satisfactory, as the electrocoagulation progresses under direct transantral exposure, gentle traction on the diathermy electrode, which tends to adhere to the tumor, frequently will bring the encapsulated surface of the mass into view, and thereby assist greatly in its clean removal. At times also blunt dissection is helpful in this connection (see Fig. 5). These measures are followed by packing radium tubes or needles into the wound, even though removal appears to be complete, or by implantation of radium needles or radon seeds into any recognizable remnant of fibroma when complete surgical removal cannot be accomplished. In some of the cases taken care of in this manner it is possible to eradicate the tumor in a single operation, which rarely is feasible when an extensive active fibroma is approached through the nose and nasopharynx; however,

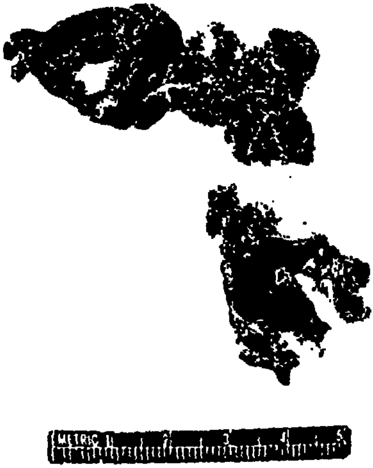

Fig. 5. Juvenile basal fibroma of the right nasal fossa, nasopharynx, sphenoid sinus and retromaxillary fossa, which was removed through a transantral approach. The tumor had a broad sessile attachment on the posterior portion of the lateral wall of the nasal fossa.

even with adequate exposure complete removal in one operation may not be possible, and residual tumor or further growth of a remnant of it must be taken care of later.

While active bleeding commonly occurs from fibromas of the nose and nasopharynx, severe secondary hemorrhage from the operative wound does not take place as often following removal by diathermy as one might well expect. Bleeding sufficiently severe to necessitate ligation of the external carotid artery was encountered in only two cases in this series. In view of this, we do not deem it advisable to perform ligation of the external carotid artery routinely as a preliminary measure. Ligation of the external carotid artery on the side of the principal involvement was carried out immediately preceding transantral exposure of the tumor in five cases in this series. It was our impression that this was of material assistance in controlling the hemorrhage, although bleeding was profuse in some cases, in spite of this procedure. Bilateral ligation of the external carotid arteries probably would have helped considerably more, and one need have no hesitancy in carrying this out when it is indicated. When ligation is performed, it should be at the same time as the actual removal of the growth in order to be most effective. Collateral circulation often is established so promptly that the lapse of a few days is likely to lessen its effectiveness greatly. It probably would be of no permanent benefit in preventing the severe bleeding at times associated with the crusting resulting from treatment.

Secondary operations usually are best carried out after an interval of six weeks to two months, but in a few instances we have performed the second stage of an operation after approximately two weeks. This was required in two of the cases in this group. One of these was rendered necessary by the fact that the patient reacted badly during the initial operation, the pulse becoming very rapid and barely palpable, and there being no response to a transfusion given on the table. Another patient requiring a two-stage removal presented an extensive, partially necrotic tumor filling the right nasal fossa, antrum, ethmoid labyrinth and sphenoid sinus, and extending into the retromaxillary fossa and nasopharynx. With the exception of its retromaxillary portion, this rapidly growing fibroma appeared to have been completely removed at the first operation (see Figs. 6A and 6B). Since there had been a good deal of bleeding during the procedure, and the operation had been rather protracted, it seemed inadvisable to prolong it further. It was terminated, and radium points were inserted into this remnant; however, in spite of the interstitial radiation, about 10 days later what looked like actively recurring fibroma appeared in the lower posterior part of the maxillary sinus. This was explored through the antrum on the twelfth postoperative day, and an encapsulated lobulated tumor measuring 7 by 4.5 by 3 cm., which microscopically proved to be a vascular fibroma, was removed by blunt dissection, and the wound was lightly electrocoagulated (see Fig. 7). Nineteen days later there was severe bleeding from the wound, probably due to the giving way of the internal maxillary artery, and ligation of the external carotid artery became necessary. This patient, 15 years of age, is now in robust health and free from tumor after 20 months.

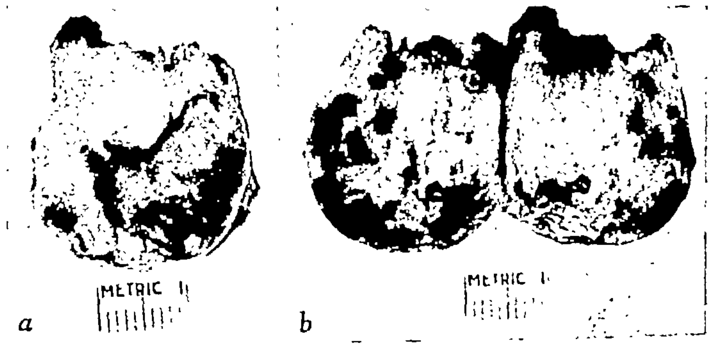

Fig. 6A. Encapsulated fibroma of the nasopharynx. This portion of the tumor was situated in the nasopharynx and dropped down into the hypopharynx upon transantral electrocoagulation of its pedicle. It comprised only a small part of the entire fibroma, which filled the right nasal fossa, antrum, ethmoid labyrinth, sphenoid sinus and nasopharynx of a lad 15 years of age. (B) Tumor opened up to show its homogeneous character.

The effectiveness of therapy for nasopharyngeal fibromas often is difficult to evaluate because so many factors must be taken into consideration. Important among these are the age

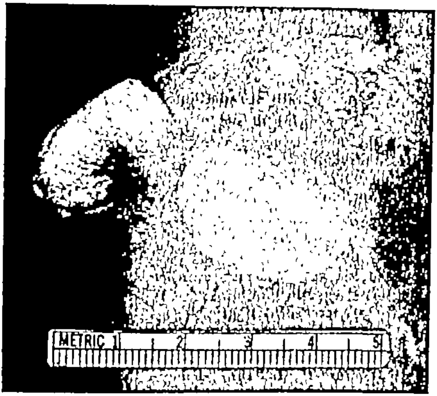

Fig. 7. Residual encapsulated lobulated fibroma removed from the posterior part of the right antrum and retromaxillary fossa 16 days after the main portion of the growth, including the mass shown in Figs. 6A and 6B was taken out. This portion of the tumor measured 7 by 4.5 by 3 cm.

of the patient, the accessibility of the tumor, its activity, size, vascularity, site of attachment, previous treatment, and its conformation; that is, whether it consists of a compact mass or of multiple lobules. Allowing for the variation in size and activity of the tumors, the length of time that therapy had to be continued and the number of treatments required in each case probably are the most accurate criteria of the potency of treatment.

Of the 51 patients in this series, one did not stay for treatment. Forty-two received electrocoagulation and interstitial radiation (see Table 3). This was applied through the nostrils and nasopharynx in 33 cases, through the antrum in nine, and through a nasofrontal approach in two. Several of these 42 patients were treated through more than one approach at different times. One patient was treated by electrocoagulation only. Seven patients received interstitial radiation only, radium needles or radon seeds being implanted through the nostrils and nasopharynx. In two of these seven

TABLE 3.
NASOPHARYNGEAL FIBROMAS. METHODS OF TREATMENT.

		Number Treated
Electrocoagulation and interstitial radiation	*	42
Nasal and nasopharyngeal approach	33	
Transantral approach	9	
Nasofrontal approach	2	
Electrocoagulation only		1
Interstitial radiation only		7
Total		50
Supplemental procedures		
Radium packs		3
Primary external carotid ligation		5
Secondary external carotid ligation		2
Total		10

*Several patients were treated through more than one approach at different times.

patients implantation of radon seeds was also made through the soft palate, and in one through the cheek, the lateral wall of the nose and directly through a bony defect in the hard palate into the mass that was perforating at this point.

In 10 cases supplemental procedures were used. In five cases primary, and in two cases secondary, ligation of the external carotid artery was performed. Three patients with extensive diffuse recurring fibromas received external radiation with radium packs in addition to the therapy mentioned previously; however, it is our feeling that radiation either with Roentgen therapy or with radium packs is much less effective in controlling these fibromas than is radium applied to the surface of the growth or implanted into it. It also bears the possible danger of inducing secondary radiation changes in the skin, and since most of the patients with nasopharyngeal fibromas are in the stage of active development, the possibility of interference with normal osseous growth is ever present. In view of the foregoing considerations, the wisdom of using intensive Roentgen therapy in these cases, as is commonly practiced, may well be questioned.

Four patients in the series received treatment less than six months prior to the time of writing of this paper, and three of these have not been observed since they were taken care of. The one patient in this group of four who has returned for observation shows no evidence of tumor, but the interval has been too brief for this to be significant. No follow-up has been obtainable for seven patients treated from four to eight years ago. The prognosis appeared very favorable for some of these, and it seems likely that at least several of them are free from tumor. One patient first treated at the age of 17 years, and who is now 20 years of age and a member of the United States Air Force, presented Roentgenographic evidence of recurrence along the wing and body of the left sphenoid bone when last observed a little more than a year ago. Treatment with radium packs only was given at that time, as no clinical evidence of tumor could be made out, and it was feared more active therapy might result in injury to the eye. The patient still has occasional attacks of epistaxis, which may be due to crusting.

Thirty-six of the 39 patients for whom a follow-up was obtainable present no evidence of active fibroma (see Table 4).

TABLE 4.
NASOPHARYNGEAL FIBROMAS. RESULTS OF TREATMENT.

Cases with treatment		50
Less than six-month follow-up		4
Lost to follow-up		7
Cases with follow-up		39*
Arrested cases		36
No evidence of tumor	29	
Inactive tumor present	7	
Deaths		2

*One case uncontrolled when last seen.

Twenty-nine of these are entirely free from evidence of tumor. The remaining seven show residual thickening or a small nubbin which has remained inactive during periods of observation ranging from six months to five years. The tumors are under control in four (57 per cent) of the seven patients treated entirely with radiation and in the one who underwent electrocoagulation only. The same is true also in 24 (73 per cent) of the 33 treated through the nostrils and nasopharynx, and in seven (78 per cent) of the nine treated through a transantral approach (see Table 5).

TABLE 5.
NASOPHARYNGEAL FIBROMAS. METHODS OF TREATMENT. COMPARISON OF RESULTS.

	Number Treated	Number Controlled
Electrocoagulation and interstitial radiation	42	31 (73.8%)
Nasal and nasopharyngeal approach	33	(24) (72.7%)
Transantral approach	9	(7) (77.7%)
Electrocoagulation only	1	1 (100%)
Interstitial radiation only	7	4 (57.1%)
Totals	50	36 (72%)

Among the patients who are now free from evidence of fibroma, the number of therapeutic measures necessary in each ranged from one to 11, the average being 2.7 (see Table 6). For the cases in which interstitial radiation only was used, the number of treatments averaged three. For the cases in which the major portion of the therapy was carried out through the nostrils and nasopharynx, the number of treatments averaged 2.9. Among those in which it was applied practically entirely transantrally, the number averaged 1.8.

TABLE 6.
NASOPHARYNGEAL FIBROMAS. COMPARISON OF PROCEDURES USED IN ARRESTED CASES.

	Number of Procedures, Average	Length of Treatment, Years, Average
Entire series	2.7	1.1
Interstitial radiation only (4 cases)	3	1.4
Nasal and nasopharyngeal approach (24 cases)	2.9	1.6
Transantral approach (7 cases)	1.8	0.5

The length of time that treatment was continued prior to complete disappearance of the tumor, or until there persisted only a small, inactive remnant, ranged from six months to five years, and averaged 1.1 years. In the cases in which radiation alone was used treatment lasted for an average of 1.4 years, and the patients averaged 17.1 years of age at its conclusion. In those in which diathermy and radium were applied through the nares and nasopharynx treatment lasted for an average of 1.6 years, and the patients also averaged 17.1 years of age at its termination. In those in which surgical measures were carried out through an antral approach treatment lasted an average of six months, and the average age of the patients was 15.5 years at the time the fibroma disappeared. Contrary to the opinion of Martin and his associates,[4] expressed in their recent communication on this subject, that aggressive measures, either by irradiation or by surgery, will never completely and permanently eradicate these neoplasms, it is our firm conviction that active treatment will cure a high percentage of them. The foregoing figures strongly bear this out.

Considering the situation of these tumors and the extent of many of them, the low mortality rate associated with their removal is gratifying. There were no immediate operative deaths in the group; however, two of the patients died a few months following operation. One of them, a boy 11 years of age, in whom a very extensive recurring tumor was removed with electrocoagulation and interstitial radiation through a nasofrontal approach, returned to the clinic three months later because of severe headaches of a week's duration, and presented evidence of meningitis, to which he succumbed 12 days later. The other, a boy 17 years of age, with a recurring fibroma filling one nasal fossa, on whom we operated three times, and to whom interstitial radiation was given on two additional occasions, had a fatal hemorrhage at home four months after his tumor was last treated with electrocoagulation and interstitial radiation. This was the only other patient in the series in whom removal of the tumor was carried out through a nasofrontal approach. This is mentioned, not because the procedure had direct bearing on the fatal outcome, but because it is indicative of the high situation of the lesion.

CONCLUSIONS.

1. The most satisfactory treatment for nasopharyngeal fibromas is a combination of electrocoagulation and interstitial radiation.

2. These measures may be used through the nares and transorally through the nasopharynx, but they are more safely and effectively applied through a transantral approach with or without preliminary ligation of the external carotid artery.

3. With proper treatment a high percentage of nasopharyngeal fibromas can be completely eradicated or reduced to a small inactive remnant; in fact, by means of electrocoagulation and interstitial radiation applied transorally complete removal often can be accomplished in a single operation.

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CONGENITAL CHOANAL ATRESIA.*

FRANCIS H. MCGOVERN, M.D.,
Danville, Va.

Judging from the increasing number of cases reported in the recent literature, congenital choanal atresia should be considered an unusual rather than a rare developmental anomaly. Current interest has been chiefly centered on the surgical methods of correction of the atresia, and little attention has been paid to several important features in the clinical picture. This report of additional cases of choanal atresia is made to review the effect of unilateral and bilateral total choanal occlusion on the physiology and development of the nose, sinuses and ears and to comment upon the hereditary aspects of this condition.

My experience comprises five cases of total unilateral bony occlusion of the posterior nares and one case of bilateral choanal atresia in an adult. A recent case was the infant daughter of a woman whose occlusion I had corrected five years previously. This was an unusual finding of unilateral congenital choanal atresia in two generations.

Little is noted in the literature regarding the hereditary nature of congenital choanal atresia. It is usually stated that the condition is rare, has little predisposition for associated congenital defects and has a low hereditary incidence. A survey of the available references has shown that Stewart[1] reported bilateral atresias in sisters and a history of an infant brother who died of asphyxia in infancy, probably due to the same cause. Wilkinson[2] cited Lang reporting a boy, his sister and mother, and possibly a brother and a maternal aunt of the

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 25, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

mother with unilateral atresia. Phelps[3] states that there is on record a family in which the mother, her two sisters, her two daughters and a son were affected. Wilkinson[2] reported complete bilateral bony atresia in three sisters. In discussing this paper, Shea stated that Guyton had two cases, cousins, under his care. Evans[4] reported a case of bilateral bony occlusion of the choanae in a three weeks old infant. Four other children in this family died in early infancy. The history strongly suggests the presence of the same deformity. In a recent communication, Dr. J. L. Cabaniss, of Roanoke, Va., reports an interesting family under his care, consisting of a mother and twins. The mother has complete choanal atresia on the left side; one of the twins has a complete atresia on the left side and partial on the right side, and the other twin had bilateral choanal atresia operated upon by Dr. Fletcher Woodward at the University of Virginia.

Macklin,[5] after reviewing this data, states that the finding of a rare developmental condition in several members of the same family bespeaks a genetic origin; however, she points out, "the mode of transmission is not easily answered. The few families mentioned in the literature would indicate that it is a dominant factor in most instances."

The most interesting, but infrequently discussed, features of congenital choanal atresia are its relationship to the development of the sinuses, and to the physiology of the nose and ears. To illustrate how little the development and function of the nose and sinuses are affected, the following cases, one unilateral and one bilateral, are reported as characteristic of choanal atresia in an adult.

The patient, a woman age 52, stated that she had never been able to breathe or smell through the left side of her nose. It had caused her little inconvenience; occasionally a mass of mucus would drain from the left nostril, especially on flexion of her head. Her ears have never given her any trouble.

Examination showed a complete bony atresia of the left posterior nares. The mucous membrane was pale and boggy and responded fairly promptly to the application of vasoconstrictors. The left nasal fossa was narrowed by a deviation of the septum to the left. A moderate amount of glairy, tenacious mucus was removed from the left fossa by suction. Most of the mucus was collected in a depressed recess formed in the anterior portion of the floor of the nose. Anosmia was present on the occluded side; the olfactory sense appeared slightly impaired on the opposite side. The skin of the upper lip on the left side showed areas of excoriation. The right nasal fossa appeared normal. No other abnormalities or asymmetries were noted.

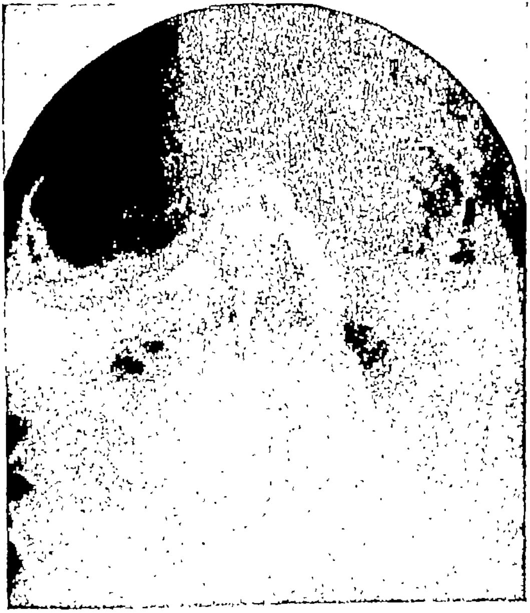

Fig. 1. Unilateral choanal atresia. X-ray of sinuses demonstrating full development of maxillary and frontal sinuses.

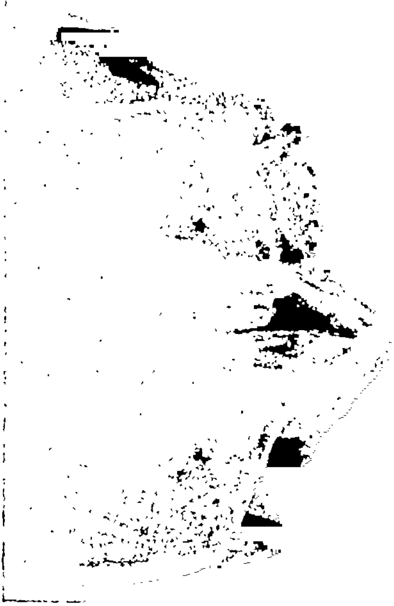

Fig. 2A. Lipiodol filling left nasal fossa, demonstrating occlusion of left posterior choana.

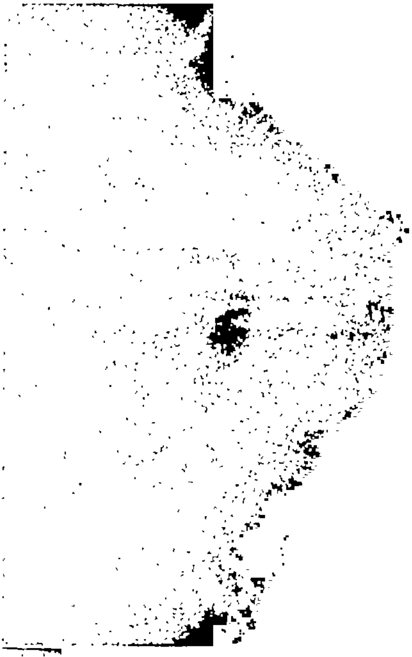

Fig. 2. Case 1. Unilateral choanal atresia. Lipiodol in left nasal fossa.

The X-ray examination of the sinuses (see Fig. 1) was negative except for the thickening of the lining of the left maxillary sinus; all the sinuses were fully developed. The left antrum was clear on irrigation. Fig. 2 illustrates the filling of the left nasal fossa with lipiodol and occlusion of the left posterior choana.

Powder blown into the nasal fossa on each side was promptly removed posteriorly by ciliary action. The ciliary movement appeared exceedingly fast on the obstructed side. A section of mucous membrane was removed from the anterior tip of the inferior turbinate for microscopic examination and was reported as follows (see Fig. 3): "The sections show

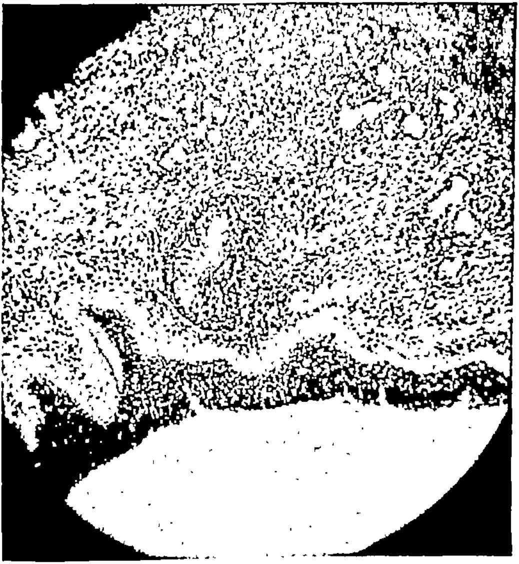

Fig. 3. Congenital choanal atresia. Photomicrograph tissue anterior portion of lower turbinate.

several collections of polymorphonuclear leucocytes in the fibrous tissue beneath the mucous membrane and a sprinkling of acute and chronic inflammatory cells throughout the fibrous tissue. The tissue is lined by normal appearing pseudostratified, ciliated, columnar epithelium. Aside from the acute and chronic inflammatory cells, the appearance is essentially that of normal nasal mucosa."

The tympanic membranes were dull and slightly retracted. The audiogram showed a bilateral symmetrical perceptive type of hearing impairment (see Fig. 4). The nasopharynx, except for the occlusion of the posterior nares on the left side, did not appear unusual.

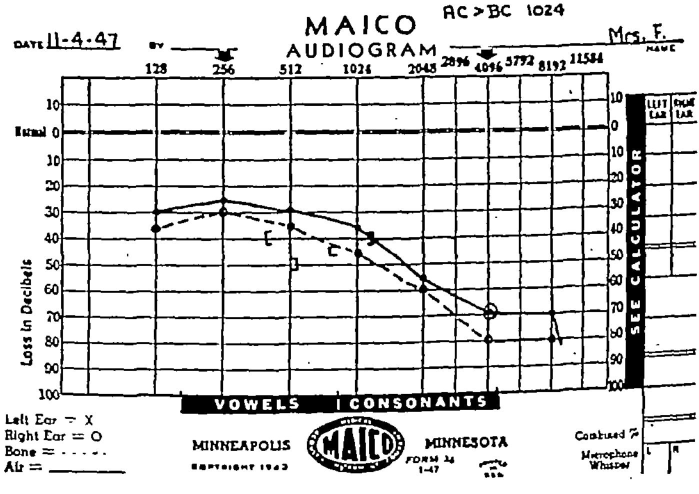

Fig. 4. Audiogram, Case 1.

The stained smear of nasal mucus showed many polymorphonuclear leucocytes but no eosinophiles.

The second patient, age 19, had never breathed through her nose. She had complete anosmia. Except for wiping her nose frequently, this disability has never given her any particular trouble. She thinks she has more "head colds" than the average person. Her hearing has been adequate and her ears have never bothered her. As a baby, she heard her mother say she was frail and had some difficulty in feeding; however, as long as she remembers she has never been sick. She is now the mother of a healthy baby girl. There was no family history of choanal atresia.

Examination showed a slender, attractive young lady. There was no facial asymmetry; the teeth were normal, and no palatal deformity was present. The tonsils were atrophic; the nasopharynx appeared normal except for the complete occlusion of each choana. The nasal mucous membranes appeared boggy with a bluish cast. The lower and middle turbinates were slightly atrophic and because of widening and lowering

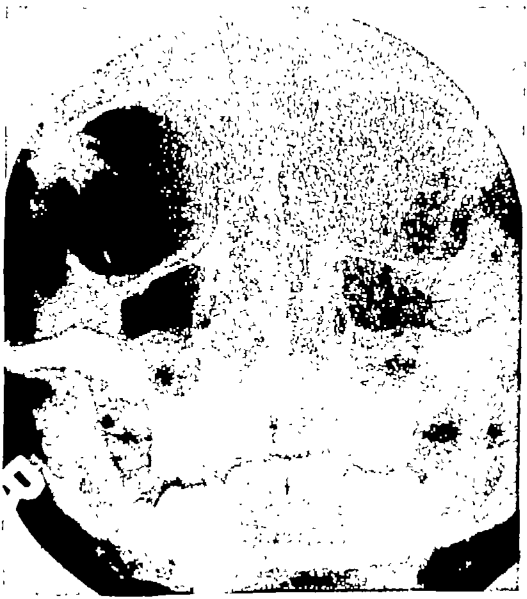

Fig. 5. Bilateral congenital choanal atresia. X-ray of sinuses.

of each nasal fossa and inferior meatus, appeared highly placed. The recessed nasal floors were filled with thin, glairy mucus. The inferior edge of each lower turbinate presented a slight mulberry degeneration. The posterior end of each fossa was completely occluded by a solid bony partition.

X-ray of the sinuses (see Fig. 5) showed small frontals especially on the right side. The maxillary, ethmoid and sphenoid sinuses were well developed; all of the sinuses were clear. Fig. 6 illustrates each nasal fossa filled with lipiodol.

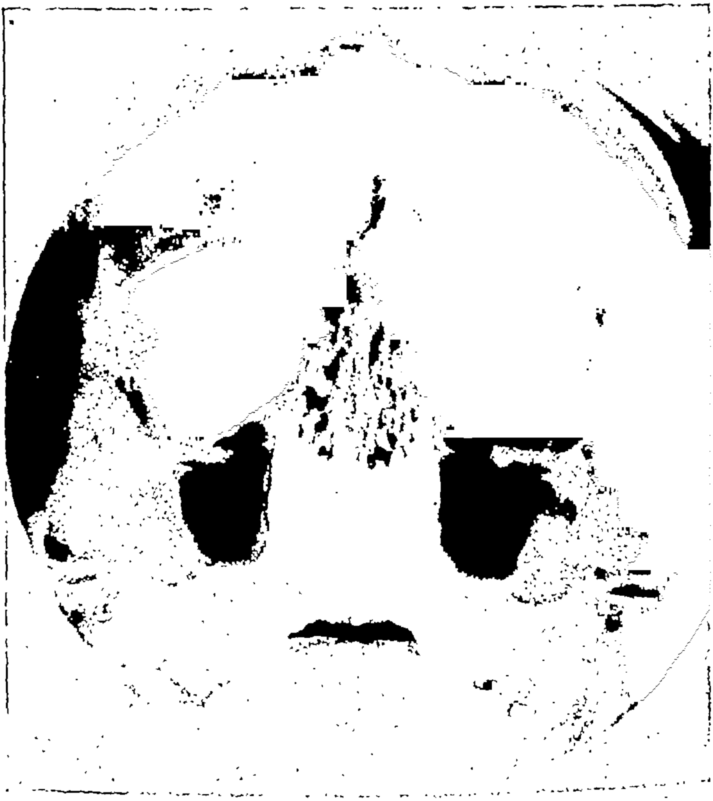

Fig. 6A. Bilateral congenital choanal atresia. Lipiodol study.

The tympanic membranes appeared slightly retracted; audiometric examination showed normal hearing (see Fig. 7).

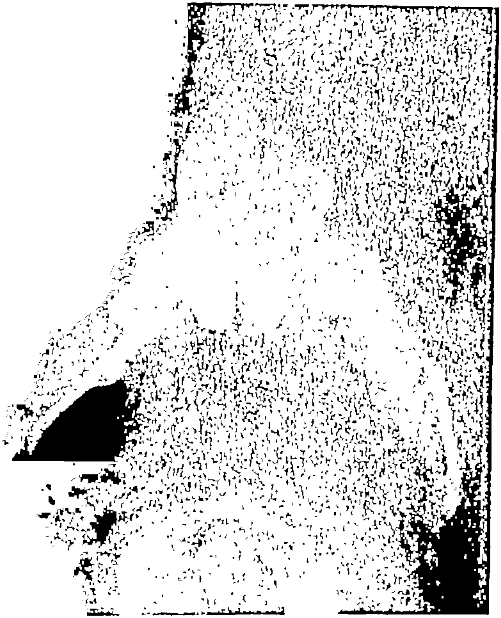

Fig. 6B. Bilateral congenital choanal atresia. Lipiodol in each nasal fossa.

Powder placed in each nasal fossa was rapidly carried posteriorly by active ciliary action. There appeared to be a triangular area of stasis at the anterior tip and along the inferior edge of each lower turbinate.

The only associated abnormality was a webbing of the second and third toes of each foot. General physical and laboratory examination was negative.

COMMENT.

In the majority of articles on congenital occlusion of the posterior nares the anatomic lesion and operative correction is

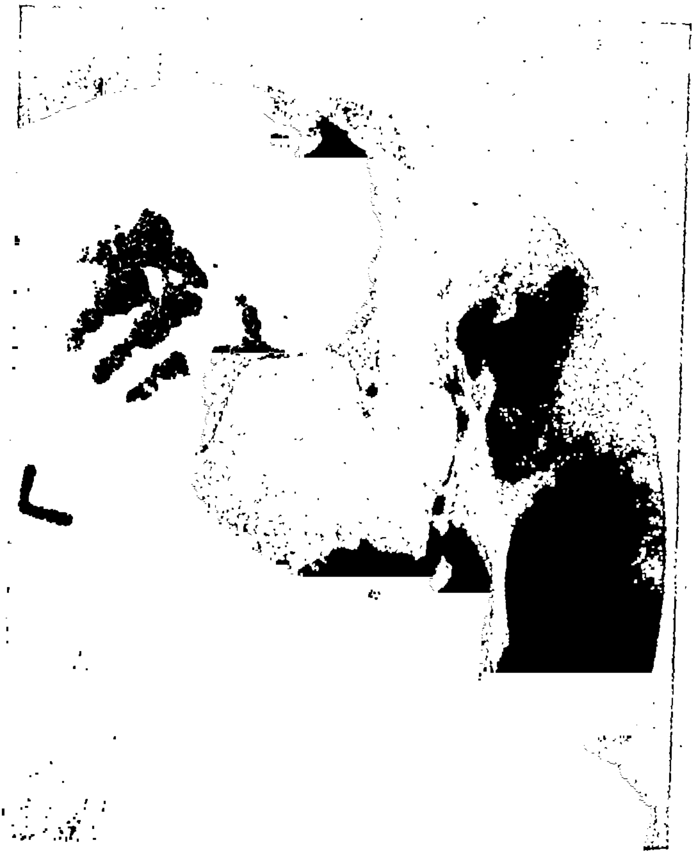

Fig. 6C. Bilateral congenital choanal atresia. Lateral X-ray with lipiodol.

described, but little mention is made of the anatomic development or disease of the sinuses. It can be assumed that these structures were normal in most instances. In the past it has been stated that all cases of choanal occlusion would sooner or later develop sinusitis, although the normal development of the sinuses was not impaired. Proetz[6] said, "It is generally agreed that stagnation of mucus in the closed side is apt to result ultimately in sinus disease." Grove[7] stated that, due to lack of air currents and free ventilation plus impaired ciliary action, the sinuses on the affected side were prone to develop sinusitis. Dean[24] found all children whom he had examined with choanal atresia had sinusitis.

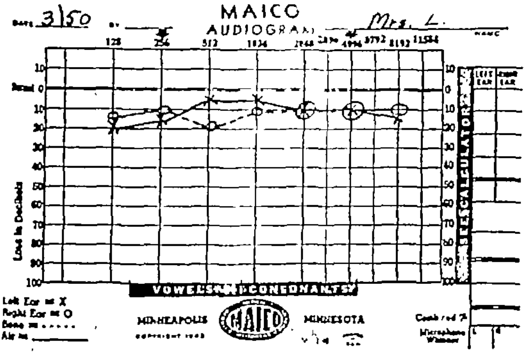

Fig. 7. Audiogram, Case 6.

A review of the cases of choanal atresia reported more in detail in the literature indicates that the anatomic development of the sinuses, in either bilateral or unilateral atresia, is unimpaired, and that sinusitis is an occasional rather than a routine finding. In 47 cases reported by 25 observers, 46 showed clinical or X-ray evidence of normal development of the paranasal sinuses. In one case the frontal sinus on the occluded side was underdeveloped. Four of the 47 cases showed evidence of sinusitis.

A similar analysis of the cases of congenital atresia reported in the literature indicates that aural involvement is not frequent. It is usually reported that the drums are thickened, dull or retracted, but the hearing is unaffected; in all but 10 of the 47 cases the hearing was essentially normal. Chronic suppurative otitis media was recorded in six cases. In one case the hearing was improved following correction of the atresia.

Regarding associated abnormalities, 10 of the 47 cases showed high arching of the palate, five cases showed asymmetry of the face, and one each showed bifid uvula, congenital coloboma of the iris, double tragus on each side, double congenital aural fistula, polydactylism, and flat nose with separation of the cartilages in the tip.

A summary of the various abnormal intranasal findings described by various observers includes: nasal mucosa pale and boggy and inelastic, polypoid degeneration of the entire mucosa, advanced mulberry degeneration, hypertrophic inferior turbinates, deviation or thickening of septum to obstructed side, turbinates atrophic and underdeveloped, turbinates short and attached to occluding plate and mulberry hypertrophy of lower turbinate.

Regarding the abnormal intranasal lesions associated with choanal atresia, Proetz[6] states that "a difference apparently exists between the behavior of a nasal mucosa deprived of air after breathing is established, and one in which, through congenital posterior closure, no breathing has ever taken place. In the latter condition normal, healthy tissues are often found in the nasal fossa, and there is also an absence of the hyperphasias and similar reactions usually encountered about the orifices of the Eustachian tubes in cases of acquired obstruction." Thomson[8] remarks, "It is noteworthy that this complete atresia of the nose, even with entire mouth breathing, does not entail the chest deformity, narrow nostrils, alar collapse, deflection of the septum, and changes in the ears which are frequently attributable to partial stenosis, or to chronic nasal infection, or to both." The most significant feature, therefore, is the difference between complete and partial nasal obstruction.

Except for the observation by Shambaugh[9] no mention has been found of the effect of choanal occlusion on ciliary function. Shambaugh studied the ciliary action after operation and reported normal function. In the cases here reported powder blown into each nasal fossa was quickly swept backwards, equally well on obstructed and unobstructed side.

Similarly, the sense of olfaction may be unaffected by choanal atresia; after surgical correction of the obstruction the sense of smell was restored in four of the 47 cases.

The few biopsies of nasal mucosa in cases of choanal occlusion have been reported in the literature and these have been significantly negative; generally there is little deviation for the normal nasal mucous membrane. The biopsy taken, as mentioned in Case 1, is in agreement with this finding, with the exception of the presence of ciliated columnar epithelium at a site where usually stratified squamous epithelium is found.

It is interesting at this time to compare the condition of the nasal mucosa in the laryngectomized patient and that seen in choanal occlusion. As reported by Dixon,[24] the color of the nasal mucosa after laryngectomy turns from a pink to a light purple, similar to the color of the allergic mucosa but without bogginess. The ciliary function appeared normal and the olfactory sense unimpaired, although the patients stated that the sense of smell was almost lost. The nasal mucosa was found uniformly moist; the dry, inactive, nonciliated areas became active. These patients did not have a postnasal discharge, and rarely had an upper respiratory infection. The microscopic examination of tissue removed from the anterior tip of the lower turbinate showed ciliated pseudostratified columnar epithelium in contrast to the stratified squamous epithelium seen in four out of five controls.

In Fig. 9 a summary of my six cases is made.

The treatment of congenital choanal occlusion is surgical. In the small child or infant the transpalatine approach[10] is advocated. Uusually the intranasal operation is used, with or without a submucous resection and removal of the posterior end of the nasal septum. If sufficient space is present the obstruction is adequately removed by direct approach, through the nares by means of perforating trocar and biting forceps and punches. Electrocoagulation has been reported by Morgenstern[11] as an effective simple office technique in adult cases, especially if the bone is thin. Wilkinson[2] and others advocated the use of a long shanked dental burr. Successful approach to the atresia has been made in suitable cases via the transantral and trans-septal[12] route.

Fig. 9.

Case	Age	Type	Operation	Sinus, Ear or Associated Abnormalities
1	52	Unilateral—left	Refused	Bilateral nerve deafness not attributed to choanal atresia
2	20	Unilateral—right	Successful transnasal	None
3	45	Unilateral—right	Refused	None
4	15	Unilateral—left	Successful transnasal	None
5	Newborn	Unilateral—left	Deferred	None
6	19	Bilateral	Successful transnasal	Syndactylism

Many ingenious methods have been devised to maintain the patency of the choana postoperatively. Rubber and metal tubes have been found irritating and often lead to stenosis rather than prevent it. Gauze packing has many disadvantages. The most successful obturator is one made of acrylic.

SUMMARY.

The development and physiology of the nose and paranasal sinuses is apparently little affected by total unilateral and bilateral choanal occlusion. Many reports have been made and X-ray studies have been exhibited to illustrate complete development of the sinuses in cases of choanal atresia. Biopsy studies of the nasal mucosa have shown in the cases here reported, and in other recorded cases, no departure from the normal structure. The physiological activity of the nasal cilia appear to be unaltered by the presence of this congenital anomaly. These findings suggest, as Boies has stated in the discussion of Wilkinson's[2] paper, "that nasal respiration is not important to the development of the sinuses" nor, it would seem, to the normal physiology of the nose.

Sinusitis is an uncommon rather than a common complication of congenital choanal atresia. These patients are not prone to acute infections of the nose and sinuses; the incidence of acute rhinitis is less than average. The history of continuous "colds" is a result of the periodic drainage of the retained secretions.

The finding of aural disease and hearing impairment associated with choanal atresia is not common. Middle ear disease, if present, is often found on the unobstructed side. The audiogram is usually reported as normal. The tympanic membranes are usually described as dull, lusterless, thickened or retracted. The interesting audiogram in Case 1 shows a bilateral perceptive type of impaired hearing. Fig. 8[3] illustrates an essentially normal audiogram of a 45-year-old colored woman (Case 3) with total right posterior nares occlusion. Wilkinson[1] reported improvement in hearing in one case after surgical correction of the occlusion.

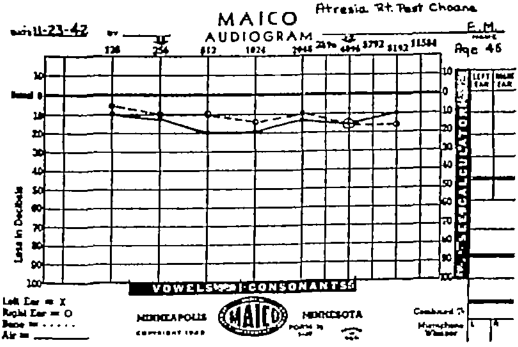

Fig. 8. Audiogram. Case 3.

Adult patients with unilateral or bilateral choanal occlusion, as a rule, complain of little discomfort or distress because of their disability; surgical correction is often refused. The accumulation of the thick mucus is removed by the patient by gravity, the patient lowering the head and permitting the retained material to drain, assisted by pressure on the ala of the nose.

Bilateral choanal atresia in infants is of grave importance and requires prompt surgical correction; failure to recognize this condition has resulted in the death of infants within the first week of life, under the mistaken diagnosis of asphyxia neonatorium. Thompson[8] states that "the nose is the natural and instinctive channel for carrying on respiration. With rare exceptions all infants breathe entirely through the nose. If the nose of a sleeping child is gently closed, he will continue to make increasingly violent efforts to draw through it; when this proves useless, he will waken and gasp for breath, instead of simply opening and using the mouth as an airway. Even when mouth breathing is partially adopted to supplement the nasal airway, the latter remains the route preferred by instinct, and as much air as possible is drawn through it. Mouth breathing is an acquired habit." The diagnosis is suspected if the infant is unable to nurse, or has spells of suffocation and cyanosis which are relieved by crying and aggravated when nursing or sleeping. If the mouth breathing habit is established, the infant usually proceeds to normal growth and development except for the nasal obstruction and associated abnormalities. Unilateral atresia in the infant is largely a feeding problem and the prevention of malnutrition. Surgical correction may be undertaken in later life.

CONCLUSION.

Congenital choanal atresia is an unusual but not rare rhinological anomaly. The finding of the same defect in a mother and daughter suggests a definite hereditary tendency. Complete bilateral or unilateral atresia has surprisingly little effect on the development of the paranasal sinuses or on the physiology of the nose or ears. The association of sinusitis and choanal occlusion is uncommon. Aural disease and impaired hearing is infrequently seen in choanal occlusion.

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BILATERAL ABDUCTOR PARALYSIS. THE POSTOPERATIVE CARE AND SPEECH THERAPY FOLLOWING ARYTENOIDECTOMY.*†

De Graaf Woodman, M.D., and
Doreen Pollack, L.C.S.T. (by invitation),
New York, N. Y.

The postoperative care of bilateral abductor paralysis may be divided into surgical or medical problems and the rehabilitation of the patient's voice.

First let us consider the immediate care following surgery.[1,2,3] A drain has been placed and held in position by a skin suture. A snug pressure type dressing has been applied. Special nursing should be provided the first 24 hours to insure frequent suctioning of the tracheotomy tube. Coughing should be discouraged and no attempt made on the part of the patient to talk or whisper the first week. In those cases which have not had the benefit of a previously placed tracheotomy tube, sedation against coughing is generally needed. These are the cases in which coughing and undue strain might help to dislodge the lateralized cord. Unless there is some economic or other very good reason it is better to perform a two-stage procedure and have the tracheotomy tube placed before doing the arytenoidectomy. Fortunately, most of the cases come to us with the tube already installed. Such patients can be much more satisfactorily prepared medically as to their thyroid and parathyroid balance if the tube is in place, relieving them of their dyspnea. A recent article by Murtagh[4] has graphically demonstrated the results of improvement in ventilation and the return to normal of the red cell indices following relief of the marked dyspnea characteristic of these cases.

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*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 26, 1950.

†From the Department of Otolaryngology, Presbyterian Hospital, College of Physicians and Surgeons, Columbia University, New York, N. Y.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 5, 1950.

If fluids are not taken readily by mouth the first postoperative day they are supported with infusions. It is rare that this is needed after the first 48 hours. We have not needed the aid of a feeding tube, though there is no reason why it cannot be used. Soft diet is generally tolerated after the third day.

All patients are under antibiotic therapy. We have used either sulfadiazine or crystacillin This is continued until the wound is completely healed. It is not unusual to have a temperature of 101° the first postoperative day, which generally subsides to normal thereafter.

The drain is half pulled in 48 hours and completely removed on the third postoperative day. The stitches are half removed on the fifth day and all out on the seventh postoperative day.

The patient is urged to get out of bed the day following operation for a short period and in gradual amounts thereafter. Most of them are up and around after the third day.

No attempt is made to inspect the larynx with mirror until the third or fourth day. Little will be seen of either cord until the end of the first week. The amount of edema may then be checked regularly with the mirror and when the edema has subsided sufficiently and the glottic chink is adequate, tracheotomy tube corking is started. At first we use a half cork and work up to full cork as tolerated. They are decannulated after four days of full corking with trials of vigorous exercise and the patient feels well satisfied that he or she may carry on without the tube. Our average time for decannulating is 26 days. There is no advantage in rushing this until well satisfied with the airway obtained.

MEDICAL CARE – POSTOPERATIVE.

The postoperative medical care is made much easier if we have had preoperative determinations of the blood serum calcium, phosphorus and serum cholesterol. Holinger,[6] in 1944, was the first to emphasize this need in cases undergoing arytenoid surgery. The presence of a tracheotomy tube precludes the value of a basal metabolism test.

Thyroid activity may then be ascertained by radio tracer uptake or by serum precipitable iodine levels. The tracer uptake technique, however, may be radically modified by any recent ingestion of thyroid therapy as pointed out by Werner, Quinby and Schmidt,[6] which factor may limit its use, as many of these cases have been carried along on some thyroid medication.

One should be on the alert for those patients with low thyroid activity and parathyroid deficiency. The parathyroid lack in our series of cases has given the most alarming group of symptoms such as mental depression, anxiety, carpopedal spasm, and sometimes complete blackouts with tetanic convulsions. The latter group of symptoms are at times difficult to differentiate in a patient with a hysteria even though the blood calcium levels remain within normal limits. The patient, through hysteria and fainting spells with accompanying hyperventilation may help to bring on symptoms which are difficult to distinguish between parathyroid lack and hysteria. It is well to institute the parathyroid and thyroid therapy in any case of doubt. Those cases which appeared borderline have generally responded to such therapy even though the blood chemistry tests did not indicate the need of therapy. This suggests the possibility that not infrequently our means of measuring this deficiency is not always accurate and the patient should sometimes be treated empirically. This phase of the medical care should be under the guidance of an internist who is familiar with this problem.

In a few cases we have found an accompanying allergy responsible for some dyspnea, which is checked when the allergy has been corrected and controlled.

Those cases with parathyroid deficiency are the ones in greatest need of psychic therapy. Having them talk with previously operated patients is very helpful, and gives them reassurance. In these cases the patient should never be forced to go without the tube until he is well satisfied that he can get along without it.

SPEECH THERAPY.

While arytenoidectomy and lateralization of the vocal cord is primarily done for the purpose of giving the patients sufficient airway in order that they may carry on all of their normal activities without the use of a tracheotomy tube, we are also faced with the problem of rehabilitation of the voice.

When all evidence of postoperative edema has passed and the patient is decannulated, the patient is then started on speech therapy. They have already been seen by the speech therapist, who has told them what to expect in the line of training. Their voice has been recorded preoperatively and they have usually been introduced to other patients who have gone through the same training regime. This puts them at ease and helps to overcome the first hurdle they must meet — learning to relax. Inasmuch as most of these cases are among men or women who have had thyroid problems, many of them of the toxic variety, it is usual to find them apprehensive, worried, and totally lacking in confidence after their long experience of fighting for air and trying to overcome a voice handicapped by their dyspnea.

Postoperative patients find themselves relieved of air hunger but they now find their voices changed, not as the result of gasping for air but by the operated cord, which, if properly lateralized, is now fixed. This means an additional amount of adduction has to be accomplished by the unoperated side in order to get complete approximation for proper phonation.

Following training in relaxation, correct breathing is mastered. This is done through exercises and finally the third phase, that of timing the phonation to occur during expiration, is taught. Once these three fundamentals are accomplished, then various exercises for training in pitch are developed. With improvement comes added confidence and greater ease in building up the accessory mechanism which develop a good functioning voice.

It is true that a goodly percentage of these cases will have very satisfactory voices. Not singing voices, but voices which do not fatigue and can carry on a rapid and efficient conversation. Such voices are found among those cases where there has been the least amount of lateralization. This minimal amount may be well tolerated and satisfactory in a lightweight woman who does not have to do any physical work, but for those cases which need the extra airway resulting from greater physical demands, it is imperative that they have their cords lateralized to a 5 mm. glottic chink at least. These cases are particularly helped by speech therapy. There is also a group of cases, fortunately small in number, in which there has been too much lateralization, where the glottic chink is 6 mm. or more in width. Although this latter group has a voice adequate for all the functions of living and can even be understood over the telephone, their voices are distinctly rough and they, too, can be helped by speech therapy.

In a recent article, Clerf[7] made the observation and statement that "the wider the glottis is, the poorer the voice will be." Even though lateralization of the vocal cord may be carefully checked with the laryngoscope at operation, such factors as size, shape and rigidity of the laryngeal cartilages, together with edema and scar tissue secondary to operation enter into the resultant lateralization. While experience does help, the precise amount of lateralization is not exactly predictable.

Voice therapy for cases of aphonia and dysphonia postarytenoidectomy, as reported by Doreen Pollack, voice therapist of the Vanderbilt Clinic. Among patients attending the Vanderbilt Clinic, the following *symptoms* have been observed:

1. Anxiety with accompanying nervous muscular tension, because they are unable to produce a normal voice.
2. Harmful attempts to produce a voice, ranging from attempts to speak during inhalation, to harsh forced whispering.
3. Respiratory distress, with noisy shallow mouth breathing during speech, which results in an excessively breathy

phonation and prevents the voice from being audible or sustained, and may cause a feeling of pain in the chest and throat. This type of dysphonia was described by Jackson and Jackson[8] in 1935. This group of symptoms sets up a vicious circle, because the patient is embarrassed by the harsh whisper, and attempts to make this louder by using more muscular tension, which in its turn, leads to more respiratory distress. The noisy mouth breathing is, of course, a habit formed before the operation.

4. In most cases there is insufficient closure of the glottis (postoperatively) for a normal vocalization. If a vibration of the vocal cords can be produced, it is usually gruff and weak, lacking inflection.

The *voice therapy* used is based on normal voice production. This is explained to the patient in simple terms.

1. A steady expiration of breath through the relaxation of the chest walls and diaphragm.
2. Initiation of the vocal attack with expiration and without excessive muscular tension.
3. Use of the resonating chambers of face to give carrying power and tone to the voice, thus relieving laryngeal hypertension.
4. Use of the optimum pitch.

Aims of the therapy; and a brief summary of the techniques used:

1. Strengthening and regulating the excitation of the vocal cords, or in some cases, the ventricular bands, by teaching inaudible inhalation, and a slow, steady economical exhalation, through a conscious control over the movements of lower ribs and diaphragm. It is important for the patient to keep the upper chest and shoulders firm. This control is then used in simple speaking exercises; counting, repeating phrases, and reading aloud.

2. The laryngeal muscles are strengthened and the glottis closed by various "pushing" exercises, following the principle that pressure exerted by hands and arms with a firmly held chest tends to close the glottis. (For example: the closed fists are held at the level of the nipples, and the patient pushes sharply downwards to the thighs, simultaneously saying AH or OO, and then other vowels. Pushing is then performed with voiced consonants and the vowels; single words; then phrases, and finally short sentences with only one push to initiate the sentence.) The therapist can also assist the action of the cords by finger pressure on the thyroid cartilage. Swallowing, followed immediately by vocalizing on EE, is sometimes helpful.
3. Resonance (*i.e.*, humming exercises) on nasal consonants, and the words containing nasal consonants, with the sensation that the voice is being sent into the front of the face is developed to produce a full tone, and relaxation of tension in the neck.
4. Finally, pitch exercises, including intoning on different notes, are given to increase the flexibility of the voice and to find the optimum pitch.

LENGTH OF TREATMENT.

This varies, but the following may be helpful:

1. Treatment must be regular, and at first should be given at least twice a week so that the therapist can be certain that the exercises are being practiced correctly.
2. Breathing, pushing and resonance exercises show marked results after one or two months, and the patient can then concentrate on finding the optimum pitch.
3. Some of the initial difficulties are caused by failure to keep the upper chest and shoulders firm, and to breathe deeply.
4. Success of the therapy depends upon the persistent practice done by the patient. Practice periods will be short at first, lengthening to about 20 minutes, but they must

be frequent. Should the patient neglect the practice during the first few months, a relapse will occur immediately. With regular and frequent practice periods, and real attempts to apply the practice during conversation, vocalization becomes effortless, and increased feeling of self-confidence and good muscular tone is noticeable.

5. It is worth mentioning that some patients experience greater difficulty during damp weather, and may relapse if harassed by family problems.

SUMMARY.

We have reviewed the postoperative care of cases operated upon for bilateral abductor paralysis and we have included the surgical and medical needs of these patients together with the value of speech therapy in voice rehabilitation.

A Kodachrome Sound Track Film followed demonstrating the open approach to arytenoidectomy with lateralization of the vocal cord. Views of vocal cords and voice recordings both before and after surgery were shown. This technique has been carried out by many surgeons thoughout this country, Europe and Canada. A total of over 150 cases have been reported to the author. The film emphasizes the importance of the role played by speech therapy in the rehabilitation of these cases.

Copies of this film may be obtained or rented through Sturgis-Grant Productions, 314 East 46th Street, New York 17, N. Y.

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CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Abraham Lincoln Hotel in Springfield, Ill., on Sept. 22, 23 and 24. The guest speakers will be Dr. Girolamo Bonaccolto, of New York, who will give several lectures on ophthalmology, and Dr. Bayard T. Horton, of the Mayo Clinic, who will speak on "Histaminic Cephalgia and the Treatment of Retrobulbar Neuritis with Histamine." Dr. C. L. Pannabecker and Dr. A. L. Paisley, two of the members of the Society, will also speak.

In Memoriam

GEORGE W. WALLERICH,

1880-1950.

George W. Wallerich, president of V. Mueller & Co., Chicago, Ill., died July 3 after a long illness. Mr. Wallerich, who was born on April 3, 1880, had been an eminent leader in the surgical instrument industry for more than 45 years. Forty years of this time were spent with the Chicago surgical instrument house which he joined as secretary shortly after his marriage to Vinzenz Mueller's daughter, Marie. Previously he was sales manager of the Victor X-ray Corp., during which time he was regarded as one of the country's outstanding authorities on X-ray equipment and techniques.

With his father-in-law, Mr. Wallerich threw his energy and imagination into the problems of the then infant American Surgical Trade Association. Together they founded and co-authored the *ASTA Journal* at their home at 2827 Jackson Boulevard, which served as the magazine's first offices. After business hours, Vinzenz Mueller edited the copy and George Wallerich tried to solicit enough advertising to keep the budding journal going.

As a lieutenant colonel, later a colonel, Mr. Wallerich served with the Sanitary Corps, U. S. Army, in World War I. During World War II, he was a member of the National Research Council.

In professional circles, Mr. Wallerich's name was respected throughout the world by medical men who sought his aid in the development of new surgical instruments and equipment. Much of his time was spent in working out improvements in old instruments for surgery and in developing new instruments for new techniques. Mr. Wallerich's intense interest in his work carried over into his personal life, and many of his closest friends were surgeons and hospital executives. He was, according to one of the latter, "a man of very high intellect and broader vision than the average man, a conscientious worker. . . ."

Mr. Wallerich became president of V. Mueller & Co. upon the retirement of Vinzenz Mueller. He was a member of the Presbyterian Church, the Oak Park Country Club and the Chicago Athletic Club.

Mr. Wallerich is survived by his wife, Marie Mueller Wallerich; a son, George Mueller Wallerich, vice-president of the firm; a daughter, Mrs. Constance Bruckner; and four grandchildren.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc.. 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Basic Sciences in Otolaryngology
September through June

THE
LARYNGOSCOPE.

VOL. LX SEPTEMBER, 1950. No. 9

RHINOLOGY IN CHILDREN; RESUME OF AND COMMENTS ON THE LITERATURE FOR 1949.

D. E. S. WISHART, M.D.,
Toronto, Canada.

In the journals devoted to otolaryngology the articles referring to rhinology in children continue to shrink, as will be seen from the following resumé which follows the pattern established in previous years.

GENERAL ARTICLES ON RHINOLOGY.

Furstenberg[1] with his usual clarity and sanity justifies another review of therapeutic principles and makes a plea for their more thoughtful and sounder application in a paper on antibiotics in the treatment of diseases of the ear, nose and throat. The paper should be read in its entirety. Certain of his conclusions follow.

Nearly all the acute infections of the ears, nose, sinuses and throat respond in a gratifying manner to the parenteral administration of penicillin. The isolation and identification of the causative organisms in every case of infection is an idealistic pronouncement but hardly feasible and practicable in everyday practice. When, however, a definite clinical response to the antibiotic is not promptly observed, the organism must be identified and its sensitivity determined. No other course will permit the rationalization of specific therapy.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 26, 1950.

When dealing with meningitis where time is an essential factor, delay for the identification of the organism can hardly be advocated. There can be nq compromise with the prompt institution of therapy. Antibiotics of proven values . . . should be employed in maximum dosage in conjunction with chemotherapy.

Penicillin, to be effective, must come in actual contact with the organism and maintain a sustained contact. The difficulty of applying this principle to the upper air passages is accountable, perhaps, for the disappointing results observed from the popular methods of topical administration. Penicillin aerosol is of little value in the treatment of the lower respiratory infections. It is doubtful that this technique of local application accomplishes more or even as much as the intramuscular injection of the antibiotic.

A dense capsule surrounding a chronic abscess in the neck is a barrier to penicillin therapy; likewise, necrotic tissue within the abscess renders the lesion resistant to the antibiotic. Similar factors which influence adversely the effectiveness of penicillin are found in chronic suppurative otitis media and mastoiditis, osteomyelitis of the calvarium and facial bones, and chronic nasal accessory sinus disease. These pathological entities are not cured by any antibiotic in our present therapeutic armamentarium, either when used systemically, locally, or by both methods.

An admonition seems timely in regard to the possible injurious effects of penicillin in the absence of infection. The normal basic flora of the throat may be altered to include harmful organisms. It is to be emphasized also that the prolonged administration of the antibiotic may produce resistant organisms which fail to respond to penicillin therapy at some subsequent time when the antibiotic is sorely needed. He has a staff member under his observation who has received intensive penicillin therapy for a chronic maxillary sinusitis. The treatment has been futile. More perturbing, however, is the fact that he harbors a pneumococcus in his antrum which is resistant to penicillin. This raises the question, has this patient lost an important defense against the possible future development of pneumococcic complication?

Enders[3] reviews briefly those viruses pathogenic for man which, during the course of the disease they incite, may be present in the mouth, pharynx and adjacent structures and then considers in more detail our present knowledge of three virus diseases: acute herpetic gingivostomatitis, stomatitis and diarrhea of infants, and mumps.

He finds that two misconceptions regarding viruses are somewhat widespread: *1.* that laboratories equipped for the study of viruses are prepared to attempt the isolation of virus or to carry out diagnostic serologic tests on any specimen which may be submitted for "virus studies"; and *2.* that a tentative diagnosis of "virus infection" is appropriate in conditions which appear to be infectious but in which no etiologic agent can be identified on the basis of either clinical or laboratory data.

Seventeen virus diseases are listed in which the agent may be present in oropharynx or nasal passages. Although in none of these is it possible by laboratory methods to confirm or establish the diagnosis their impracticability in the routine diagnosis of a disease as important as nonparalytic poliomyelitis as well as their complete deficiency in most of the common viral diseases of childhood must be regarded as unsatisfactory.

When the laboratory diagnosis can be made it is based either upon the isolation of the virus in a susceptible animal or upon the demonstration of the development of specific antibody during the acute phase and early convalescence. Even with those viruses that are most easily propagated in the laboratory, such as influenza, mumps and herpes simplex, the procedures are time-consuming and expensive. Accordingly, this mode of diagnosis in most instances should be employed only under special circumstances, as in the determination of the prevailing agent in epidemics or where the clinical data strongly suggest the nature of the infection.

He also points out that the serological findings nearly always provide a diagnosis only in retrospect, since recovery frequently occurs before they can be obtained.

The Journal of the American Medical Association[3] made the following comment upon the treatment of the common cold:

> Authoritative medical opinion supports the view that no substance or combination of substances available at present can be relied upon to prevent or cure the common cold. Recent surveys of products which now appear on the market reveal that many of them make claims involving the treatment or prevention of colds which are not justified by valid evidence. Advertisers exaggerate the effects which the medicaments will have upon symptoms; nevertheless, the public will continue to spend huge sums of money unnecessarily on obsolete, illogical remedies, such as rubbing remedies, salves and ointments, until it is educated to the foolishness of such practice. A number of simple agents and measures exist which families can be taught to employ for temporary relief and from which little harm will result.
>
> The use of nose drops and inhalants probably ranks first among measures taken by the public to relieve colds. Nasal vasoconstrictors afford symptomatic relief for varying periods but are not curative. Liquid nasal medicaments which function on a rational physiologic basis are compatible with ciliary activity, do not vary greatly in their pH (5.5 to 6.5) from that of normal nasal secretions, are isotonic and are noninjurious and nontoxic. Prolonged and excessive use of nose drops and volatile inhalers occasionally results in secondary rebound congestion of the nasal mucous membranes. Prolonged administration of silver salts can produce argyrosis, while the continued use of nasal preparations containing mineral oil sometimes leads to the development of lipid pneumonia.
>
> Although popular with the public, gargling has little therapeutic utility. Ordinarily, gargles do not provide medication behind the anterior pillars of the fauces. A number of investigators find that gargled materials will not pass beyond the

third molar tooth, thereby reaching neither the tonsillar nor pharyngeal areas, because the act of gargling produces a restrictive influence. In most cases of acute, uncomplicated pharyngitis, a reasonable amount of relief may be obtained by irrigating the throat with hot, isotonic solution of sodium chloride by means of syringes or modifications of a douching apparatus.

Acetylsalicylic acid does not have any influence upon the infectious process of the common cold. Symptomatic relief of headache, fever and muscle pain is, nevertheless, of value as long as the drug does not disguise the need for more vigorous treatment. A false sense of improvement may occasionally occur, thereby permitting the patient to resume activity too soon. Numerous cough mixtures to which laymen resort without the advice of a physician are of doubtful value. Expectorants or respiratory sedatives are best prescribed by the physician, as the preparation of the cough mixture should be governed by the type of cough and the age of the patient.

From time to time claims are advanced for special diets for either the prevention or the cure of colds. The dietary measures most frequently recommended for the prevention of colds include a high protein diet, a diet low in carbohydrates and large quantities of citrus foods, presumably to establish "alkalinization." None of these theories is supported by carefully controlled studies. The supplying of extra vitamins and other food elements to the person who does not show signs of deficiency disease does not protect him against colds. Procedures which have as their objective the conditioning of the body to sudden changes in external temperature of the skin, such as cold and hot shower baths, do not lower the incidence of the common cold. The scientific evidence against the value of oral cold vaccines is overwhelming.

The treatment of the common cold consists mainly in the relief of symptoms as they arise. Treatment should never be standardized but should be suited to the needs of each patient. During the earliest stages of the common cold the prime objective is to supply moisture by means of steam inhalations to the stricken upper respiratory passages. Alcohol has been utilized for generations to abort impending colds or to treat them. In reasonable doses it causes peripheral vasodilatation and re-establishes circulation in chilled cutaneous and mucosal surfaces. Little attention need be paid to fluids in the average uncomplicated cold; it is probably best to let thirst to a great extent determine the fluid intake. Cathartics and laxatives, long employed as home remedies for colds, do not have particular value; their excessive use may lead to dehydration. Rest in bed, especially if fever is present, diminishes the severity of the common cold, limits its spread to others and reduces the frequency of complications. It has stood the rigid test of time as a most sane and effective measure.

The Council on Pharmacy and Chemistry of the American Medical Association[4] at a recent meeting (Autumn, 1949) warned against the indiscriminate use of antihistaminic substances which are now being promoted for the prevention of colds and even for the treatment of those suffering from colds. The Council, while recognizing that data have been accumulated relative to such uses, is not convinced that they are sufficient to warrant the positive statements that are being made. The Council warns that instances have been reported of users of these drugs becoming drowsy and even falling asleep while at work and, in occasional cases, while driving cars or operating machinery. A review of the present status of these products will be prepared so that physicians who prescribe the drugs may be aware of their possibilities. In the meantime the Council declares that experience with these substances is insufficient to permit knowledge of whether they are harmless when used over long periods of time; furthermore, the amounts taken in persistent colds may exceed what has been established as normally safe.

TREATMENT OF ACCESSORY SINUS DISEASE.

De Boissière[5] very rightly observes that it is in young children suffering from sinusitis that one's therapeutic ingenuity is taxed to the limit. He has treated 350 cases and used 75 other cases as controls. In no case was it necessary to resort to operation. Freedom from the concomitant symptoms was early and sustained, and no harmful results to the mucous membrane of the nose were noted. Recurrences have been infrequent, although a few have occured, but in these symptoms have been less severe than at the initial infection.

The methods he has found most satisfactory in the elimination of sinus infection are twofold: *1.* X-radiation; and *2.* Proetz displacement with penicillin 1:200 in physiological saline; in addition, the instillation of a vasoconstrictor in an aqueous physiological solution and instilled by the Parkinson's method.

Puffiness under the eyes in children is often an accompaniment of sinusitis. This fact, however, is not as widely recognized as it should be. A correspondent wrote to *The Journal of the American Medical Association* inquiring into the significance of this sign and received a reply[n] which is abbreviated as follows:

"In the diagnosis of this group of children, whose main symptom is a puffiness of the face, particularly around the eyes, there are a number of considerations which might be of aid. One might ask whether this puffiness is hereditary and whether other members of the family evidence the same symptom. This type of puffiness, of course, would not be outgrown."

While the author of this inquiry may be describing a new clinical entity, it is more probable that the puffiness noted in children in this age group is due to respiratory infections and secondary anemia. These children outgrow this condition. It is a common observation that some small children, particularly those from three to eight years of age, seem to suffer an interminable number of respiratory infections, particularly during the winter months while they are at school. These infections produce a secondary anemia of a moderate nature, and a constant inflammation of the throat and nasal passages makes their faces appear puffy under the eyes. At about eight years of age or thereafter, these children seem to acquire an increased immunity or resistance; they have less frequent respiratory infections and outgrow puffiness of the face.

That the spirit of the children is satisfactory, as are their school records, is an important differential point, for were any severe systemic illness causing the puffiness of the face as described, the children would act chronically ill and their school record would suffer.

Further diagnostic measures do not seem to be called for as the condition is self-limited.

The above answer evoked the following abridged comment:[7]

The answer to the inquiry regarding puffiness under eyes in children did not mention chronic sinusitis, the probable cause in most cases in the northern part of the United States. This is particularly true in the absence of positive urinary findings; furthermore, the associated symptoms mentioned, such as anorexia, secondary anemia and frequent colds, are common in chronic sinusitis.

Colds and puffs under the eyes were the rule in cold-susceptible children in the chest clinic and in children with rheumatic fever. In 100 rheumatic children, all of whom had chronic sinusitis, the correspondent and his co-workers had reported that 91 per cent had circles under the eyes and many had puffiness as well, particularly upon arising. The sinuses fill up during recumbency, particularly if prolonged, and produce a congestion under the eyes and beside the nose.

The Journal of the American Medical Association[8] shortly described the Proetz treatment for sinusitis for an inquiring correspondent as follows:

The Proetz displacement therapy for sinusitis consists of placing the patient's head in a hyperextended position and then filling the nasal passages with isotonic sodium chloride solution containing a dilute shrinking agent, such as 0.12 per cent phenylephrine hydrochloride or a 0.25 per cent ephedrine, and applying suction while the patient closes his palate. The suction should be intermittent in order gradually to evacuate the air and the secretion from the sinuses, thus allowing the solution to enter.

The treatment is of value in certain cases of chronic sinusitis. It is contraindicated in acute sinusitis, where it may result in spreading the infection from infected sinuses to uninfected sinuses.

Hynes[9] hopes to prove that penicillin aerosol has a place in the treatment of sinusitis. The apparatus he uses is that of Barach. The technique consists of having the patient take three or four breaths of the penicillin vapor, followed by suction (60 mm. of mercury), alternating in this way until the penicillin is entirely vaporized. A small amount of saline is then added to obtain as much of the penicillin left in the apparatus as possible. The principle involved is to obtain a negative pressure in the sinuses so that the aerosol will enter when inhaled under slight pressure. Altogether, 212 patients were treated and of these 58 were classified as clinical cures. A number of his very young patients have been treated. Most of these had been treated previously for "sinus," and had had tonsillectomy and/or adenoidectomy. Many had had a few (two to 10 tests) for "allergy." Outside of two entirely uncooperative children, every one showed marked improvement with occasional recurrence.

Winborn[10] discusses frontal sinus infections, their complications and management, and reports five cases, two of which were in children 15 and 13 years of age, respectively. He contends that extensive surgery is not necessary and that mortality is low in frontal sinus complications when these are treated conservatively in conjunction with penicillin and sulfonamides. When the quiescent stage is reached following a complication, a chronic frontal sinus should be exenterated by an external approach in order to avoid a possible recurrence.

Kler[11] states that abscess of the frontal lobe is a rare complication of sinusitis in general and an extremely rare complication of ethmoiditis in particular. In a paper on that complication he gives a description of its occurrence in a five-year-old child. The determining factors in the diagnosis were the history of an infection of the upper respiratory tract, followed by the obvious presence of a sinal infection, a periorbital abscess and signs of meningeal irritation and increased intracranial pressure. Operation revealed necrotic bone, an extradural abscess and a fistulous tract to a subcortical abscess. Recovery.

Banham[12] reports a case of osteomyelitis of the maxilla in a child of two and one-half years. Complete recovery, without the formation of sequestra, followed conservative surgery and large doses of penicillin.

About one week after damage to her left cheek from a fall her left lower eyelid was swollen, there was purulent discharge from the left side of her nose and from the sockets of two teeth which had been removed, and there was swelling of the hard palate on the left side. In spite of penicillin therapy her condition did not improve and the abscess of the hard palate was incised, the orbital abscess drained by an incision along the lower orbital margin and an intranasal antrostomy was performed. Penicillin was given three hourly for 21 days to a total of 11,000,000 units.

ALLERGY.

Grove[13] after 18 years of close contact with the allergy clinic of the Roosevelt Hospital in New York City is convinced that the importance of the disease of the sinuses in the treatment of allergic manifestations is too often underevaluated by the allergists and also by the otolaryngologists. He deprecates the pessimism of the latter and undertakes by three tables and the summary of six very complete studies of the results of his own sinus surgery in asthmatic patients to prove that excellent results can be produced by careful intelligent handling of disease in the paranasal sinuses. The cases analyzed were followed postoperatively for one-half to 17 years. The number of children in his study is not stated.

Two of his observations are noted: he does not believe that the simple washing of an antrum and finding it clear, and the report of a negative culture of the return flow indicates a normal or noninfective sinus; he uses a 25 per cent colloidal solution of thorium dioxide diluted one or two times for the demonstration by X-ray of the condition of an antrum and finds the shadows less dense and the margins and backgrounds of the sinus more clearly delineated than when lipiodol is used.

Edwards[14] presents certain aspects of a series of 148 cases of perennial allergic rhinitis and asthma. In 122 cases definite information as to the age of onset shows that this is essentially a malady commencing in middle childhood. Only two cases began under one year of age and seven under two years. In about one-half of the cases the onset dated in the age group from five to 10 years. All the patients were skin tested. Chocolate gave the greatest number of positive reactions to foods. The position of corn on this list — fourth — is of interest because it is so often used as the cereal basis for elimination diets.

Removal of tonsils and adenoids was noted in 44 cases. No relationship between the severity of the allergic manifestations and the presence or absence of the tonsils was apparent in these cases. He quotes an author as stating that tonsillectomy usually aggravates the asthma, and many children actually date their asthma from a tonsil operation; but he is convinced that children with respiratory allergy stand ether anesthesia well and feels that if tonsils are chronically diseased they should be removed, and other foci of infection cleared up in the program for improving the patient's general health.

He particularly refers to hypodermic desensitization using a mixed inhalant allergen and draws attention to the importance of the psychological management of these cases.

Levin and Moss[15] have evaluated clinically Thephorin, a new antihistaminic drug, differing structurally from previous antihistaminics, in 109 allergic children. It was found to be very effective for symptomatic relief. It produced side reactions in 20 per cent of their cases. Reactions were seldom severe enough (8 per cent) to discontinue use of the drug. Thephorin has a mild sedative effect in children in contrast to the stimulating effect seen in adult patients.

Eosinophilia is so frequently thought to be unmistakable evidence of the allergic state that a statement by *The Journal of the American Medical Association* on the significance of eosinophilia[16] is worth noting.

In general, eosinophilia is not present unless the eosinophil count is greater than 6 per cent or the total number of eosinophiles is more than 600 per cmm. A number of disorders may produce mild eosinophilia. These disorders are practically all allergic or parasitic or consist of involvement of the bone marrow. If the eosinophile determination is greater than 20 percept, the diagnostic field is somewhat restricted. Trichinosis or arsenic poisoning are most commonly associated with such high percentages, but eosinophilic leucemia, periarteritis nodosa, or lymphosarcoma may show such percentages. An eosinophilia of less than 20 per cent may indicate any one of a great number of parasitic infestations, allergic states or even bone marrow involvement.

EFFECTS OF DRUGS.

Terrell and Hoar[17] report the successful treatment with streptomycin of four young infants suffering from hemophilus influenzae laryngotracheobronchitis. The lack of clinical characteristics in young infants which would identify the etiology of the disease is shown. It is suggested that streptomycin be given immediately to all cases of laryngotracheobronchitis occurring in young infants and continued until the throat culture reveals a nonsusceptible organism.

Amethocaine hydrochloride as a surface analgesic is increasing in popularity in Great Britain. American readers will more readily recognize this drug under one of the following names: "pantocaine," "pontocaine," "anethaine," "tetracaine." Otolaryngologists use it in the nose and throat and preparatory to bronchography and bronchoscopy.

Its advantages are: it is resistant to boiling; it is stable in more potent than procaine or cocaine; and it is relatively quick in achieving its action and provides prolonged analgesia. Although the onset of analgesia is slower than with cocaine, the effect is more persistent.

Its disadvantages are: it penetrates the tissues more rapidly than procaine and does not produce the vasoconstrictive action of cocaine. It is, therefore, essential to use it in conjunction with adrenalin, which reduces its toxicity by one-fifth.

Jackson[18] reports two toxic reactions in the use of this drug as an analgesic for bronchoscopy. In reviewing the literature he finds 12 reported deaths and many cases of severe constitutional disturbance. He shows that an adequate sensitivity test is difficult and makes valuable suggestions for the prevention and treatment of toxic reactions. His paper is recommended to all those who are in the habit of using pontocaine.

PHYSIOLOGY.

Fabricant and Perlstein[19] had an unusual opportunity to study the hydrogen-ion concentration of nasal secretion *in situ* in a group of 13 newborn infants ranging from two to eight days in age. Three consecutive readings were taken at one-half-minute intervals. For the 12 newborn infants who cried, the hydrogen-ion concentration of the nasal secretion was found to range from 6.7 to 7.2.

NASAL HEMORRHAGE.

Ogura and Senturia[20] present a long, well illustrated and tabulated study of the incidence, cause and control of nasal bleeding seen in a large hospital center where both children and adults were observed. In 14 years 48 children were hospitalized for epistaxis. Epistaxis was noted present in 50 per cent of children with active rheumatic fever or inactive rheumatic heart disease. Epistaxis in children was always anterior and septal.

TONSILS.

Anderson[21] writes for general practitioners an unusually practical paper on the acute throat. The article should be read in its entirety, but some of his remarks on treatment which aroused comment require mention: *Antitoxin*—Although in many cases treatment will be guided by the results of the bacteriological examination, it is often necessary (and in a suspected case of diphtheria essential) to start treatment before the results of such examination are known. The dose of diphtheria antitoxin ranges from 5,000 to 100,000 units. Where the disease is suspected an intramuscular dose of 8,000 units will suffice. For the late, severe case when hospital admission cannot be immediately secured a dose of the order of 50,000 units will be required, and in such an event intravenous administration is particularly desirable. Such cases are best treated in hospital, however, and the practioner will find that most fever hospitals are anxious to admit them even when the diagnosis is unconfirmed or in doubt. *Penicillin*—For the coccal infections and for Vincent's angina penicillin is the treatment of choice. It should be realized that the need is to secure an adequate local concentration of the antibiotic. The condition must not be thought of as a mere surface infection. Local therapy by lozenges and so forth is, therefore, completely inadequate, and systemic administration in high dosage is indicated. If the aqueous solution is used a four-hourly dose of 100,000 units should be injected intramuscularly; procaine penicillin may be employed to advantage here, and two or three injections of 500,000 units during the first 24 hours may be followed by a lower dosage for the remainder of the time. For all throat infections due to susceptible organisms treatment must be continued for seven days to secure the best results.

Although there is no specific treatment for infectious mononucleosis the throat infection is usually due either to streptococcus pyogenes or Vincent's infection, and penicillin will prove effective. In agranulocytic angina, also, the administration of penicillin has greatly improved the outlook. Pentnucleotide should be injected intramuscularly. During the first 24 hours three or four injections of 5 to 10 ml. may be given, and thereafter the dosage will depend upon clinical response. The administration of the antihistamine drugs might be considered, but the results have not been encouraging. In severe cases repeated small blood transfusions are valuable. The diet should be plain and contain no flavorings or adulterants, and care should be taken to avoid the use of any chemical which might have a sensitizing action. *Sulfonamides*—The administration of sulfonamides has been intentionally omitted from the treatment of the acute throat. The efficacy of these drugs has never been convincingly demonstrated in such conditions. Since they are undoubtedly toxic materials, carrying appreciable risks of sensitization, their administration is inadvisable, and in certain conditions may even be dangerous.

Finally, it must be pointed out that many mild throat infections clear rapidly and effectively without specific treatment. Gargling is so frequently indulged in by the patient before seeking advice that its continuation may be advised for its effect in cleansing the mouth. For those adults who can gargle, warm water with glycerin of thymol may also prove soothing to the throat. When pain is more severe the application of poultices to the neck, inhalations of friar's balsam, and, of particular value, the administration of aspirin powder, which is allowed to melt slowly in the mouth before swallowing—such measures are often adequate to secure rapid relief. The hasty rush for penicillin as a panacea for all cases is to be deprecated. It may do the patient little harm, but it can induce in the physician an easy optimism which may eventually cloud his whole judgment.

Various correspondents criticized Anderson's views on the sulfonamides, calling forth the following reply:[22]

"I was, of course, aware that sulfonamides were widely regarded as efficacious in tonsillitis; nevertheless, it is necessary to be clear about what the sulfonamides are expected to do. I imagine I am not exaggerating if I say that of every 100 sore throats which are severe enough to make the patient seek medical advice at least 70 of them will subside, by means of the hosts' natural defenses, within a matter of 24 to 48 hours. I am not satisfied from my own clinical observation that sulfonamides hasten this process. Further, one of my assistants, Dr. J. O. French, conducted a very carefully controlled investigation of the efficacy of sulfonamides in scarlet fever. She was unable to observe any beneficial effect upon the throat or upon the duration of pyrexia in those given sulfonamide compared with a group of patients (alternate cases) not so treated. When an infection shows a spontaneous tendency to rapid cure in a high proportion of cases one must be wary of making unqualified claims for a certain measure of treatment.

"In the second place, the clinical diagnosis of streptococcal throat makes a further difficulty. From my own clinical observation, supported by adequate bacteriology, I am far from convinced that I can diagnose streptococcal tonsillitis on sight. In a fair proportion of apparently typical cases the flora obtained on a blood-agar plate is little different from the normal, and streptococci are disappointingly absent. As a rule, however, it is not difficult to recognize the effects of streptococcal invasion, and I feel that sulfonamide should be reserved for such cases. It was for this reason that I stressed the importance of examining glands, middle ears and sinuses.

"Finally, I would like to plead for thoughtfulness in the use of all the different chemotherapeutic weapons. Coming to terms with the common pathogenic bacteria is, in this imperfect world, an essential part of growing up. The throat is by long experience well equipped to cope with most of the bacteria that meet it. The occasional mild tonsillitis may be no bad thing if, as a result, our immunity mechanisms are stimulated, for we may thereby be the better equipped to deal with later attacks. The unnecessary aborting of mild illness of no danger to the patient just for the saving of a few hours in the total duration of the infection should not too easily be regarded as a good thing. The experience of the clinician in the fever hospital leaves no doubt about the great increase in sensitization rashes. The experience of the clinician and of the pathologist leaves no doubt about the great increase in the occurrence of polyarteritis nodosa. I would be foolish to say that there was an absolute correlation between these facts and the known increase in the ingestion of sulfonamides, for, of course, there has been an increase in the ingestion of all kinds of chemicals in recent years. But such facts should make us pause before using sulfonamides routinely for trivial illnesses."

Preston,[23] discussing postoperative tonsil bleeding, gives *1.* a brief résumé of the physiology and dynamics of hemostasis; *2.* comments upon 3,260 cases of tonsillectomy to emphasize certain etiological factors; *3.* a brief survey of the diagnosis and management of hemorrhage; and *4.* a general plan of management of the operation of tonsillectomy designed to reduce postoperative tonsil bleeding.

Williams[24] reports four cases of septicemia in an estimated 30,000 tonsillectomies while he has been in charge of that work at the Bristol Royal Infirmary. That in quite large series of operations so few cases of acute septicemia occur, shows how rare is this complication. He hopes that with modern methods treatment will be more satisfactory.

Alexander and Reynolds[25] state that cerebral complications of operations on the tonsils and adenoids are rare and for that reason report abscess of the left frontal lobe of a diffuse type and without a definite capsule which occurred in a child following that operation. The boy was in normal condition at the time of operation but suddenly developed abnormal symptoms at the fifth postoperative day, death occurring on the fifteenth postoperative day.

This case is of peculiar significance as it followed tonsillectomy and adenoidectomy during the infantile paralysis season and a diagnosis of poliomyelitis was made—a reasonable conclusion considering the neurological findings. The autopsy alone disclosed the true nature of the cause of death. The case suggests the possibility that conditions diagnosed in the past by others as poliomyelitis following tonsillectomy and adenoidectomy may have been intracranial complications of different origin.

Cook and Munro-Ashman[26] describe the epidemiological and bacteriological findings in an outbreak of streptococcal tonsillitis in a residential school. The outbreak extended over two terms, and 57 out of 340 boys were attacked.

All the early cases were in the same house as the first case, a heavy nasal carrier. A nasal carrier was also associated with each of the three dormitories with the highest attack rates during the time fresh infections were occurring. In most cases, however, the dormitory did not appear to be of special importance in the spread of disease, and the significance of this is discussed.

The early detection and isolation of heavy nasal carriers before widespread dispersal of infection has occurred is emphasized.

IRRADIATION WITH THE NASOPHARYNGEAL APPLICATOR.

Dow[27] reports observations on 74 patients treated with the Army type 50 mg. radium sulfate nasopharyngeal applicator for the destruction of hypertrophic lymphoid tissue in the nasopharynx. One-third of these were children. Forty-one patients were treated by placing the applicator eight and one-half minutes to each side of the nasopharynx: 33 patients were given similar treatments 12 minutes to each side of the nasopharynx. The longer treatment was observed to have removed lymphoid tissue safely and more effectively.

Although in America many reports have been made of radon treatment to the nasopharynx, few have appeared in Great Britain. At the Royal Hospital for Sick Children in Aberdeen this form of therapy has been in operation for about three years. Collins, Thomson and Swindell[28] have made a preliminary report on 41 children who have been treated and adequately followed up.

The nasopharyngeal applicator used by the authors differs considerably from that in common use in America. A glass capillary tube 3 cm. long, containing 20 mc. of radon is inserted in a platinum tube of wall thickness 0.5 mm. The platinum tube is mounted in a brass holder which can be introduced through the nostril into the nasopharyngeal cavity.

One of these applicators is passed through each nostril in such a way that the radon containers lie parallel to each other and separated from the walls of the nasopharynx by a few millimeters at the nearest point. The applicators are left in this position for one hour.

The platinum wall of the tube is sufficiently thick to absorb the B-rays almost completely, and in consequence the tube is considered a source of pure G-radiation. Two illustrations are provided showing isodose curves superimposed upon radiographs taken in two mutually perpendicular directions of the applicators in position. The illustrations will be considered by Americans as indicating that the authors' applicator must be much more difficult to use than the usual monel metal applicator. The authors calculate the dose delivered in one hour in the neighborhood of the Eustachian cushion as about 300 r. At a depth of 1 cm. in the tissue the dose decreases to 70 r. and at a depth of 2 cm. to 30 r. They point out that the American monel metal tube containing 50 mgm. of radium sulfate in contact with the tissue delivers in six and two-thirds minutes at a depth of 1 mm. 1,520 r.

The chief dangers which may arise from irradiation therapy are:

1. Interference with the centers of ossification. They point out that the field of irradiation is very small and consider this danger likely to arise only if X-ray therapy is used.

2. Irradiation burns leading to scarring and occlusion of the Eustachian tubes. In the dose used by the authors the risk from burning is claimed to be negligible providing no attempt is made to bring the applicator into contact with the Eustachian tube itself.

3. Overdosage which may leave a wide and patulous Eustachian tube as an easy avenue for infection of the middle ear.
4. Reduction of activity of the ciliated epithelium in the nasopharynx.

The authors consider that all these dangers can be avoided by the use of small doses given at widely spaced intervals. They have judged their results more by the symptomatic improvement than by the decrease in the hypertrophy of the lymphoid tissue.

Examination of the postnasal space was made by a small postnasal mirror in the majority of cases. There was no evidence of any radiation burns nor was there any scarring of the nasopharynx. In no case was any atrophic nasopharyngitis observed.

The results obtained from radon application to lymphoid tissue in the nasopharynx are, in their opinion, superior to any that they have achieved with the use of antral lavage, nasal drops, vitamin therapy or any other forms of treatment. Radon therapy caused considerable symptomatic improvement and no harmful effects were observed.

The author of this resumé feels it necessary to comment that in the list of references appended to the article there is not one paper by the Johns Hopkins group who originated this form of treatment and that examination of the nasopharynx of children by postnasal mirror is usually useless for the determination of its condition.

Schulz and Robbins[29] presented before the huge audience attending the fifty-third annual session of the American Academy of Ophthalmology and Otolaryngology the dangers of irradiation of hypertrophied lymphoid tissue of the nasopharynx. These were illustrated by pictures of some devastating results of irradiation of various lesions of the skin which appeared many years after the treatment with doses, in some cases, less than that now advocated for the treatment of hypertrophied lymphoid tissue of the nasopharynx.

Their paper should be most carefully read by all those who are interested in the treatment of the nasopharynx by irradiation. Their summary and conclusions are quoted verbatim.

"With the renewed interest in the application of ionizing radiations to the nasopharynx of children and young adults, it seems wise to re-emphasize the fact that an agent which when properly used may prevent deafness or other serious defects also has the power to do great harm. An attempt has been made to demonstrate this latter fact by citing certain unfortunate occurrences that have followed the use of ionizing radiation in the past. In many of these, changes in normal epithelium which were permanent and progressive followed a radiation dosage that was in the approximate order of the dose now recommended for use in the nasopharynx. The facts to be emphasized, often unknown or forgotten, are that these changes may not appear for many years and that multiple doses are probably more hazardous than a single application. Fortunately, breakdown of atrophic irradiated tissue does not occur very often, and malignant degeneration with even greater rarity; but, for the unhappy individual in whom one or the other does occur, the final picture is frequently worse than the original. If the breakdown of tissue is in a location readily visualized and accessible to plastic surgery, the resulting damage is not irreparable. The nasopharynx, however, is not such a location.

"*Conclusion: 1.* The monel metal nasopharyngeal applicator, while of undenied value in the treatment of hypertrophied lymphoid tissue in certain instances, holds at the same time potential dangers.

"2. When it is used in the treatment of benign lesions, particularly in the case of children and young adults, the following suggestions are offered: *a.* The operator should determine by trial on his own arm the erythema dose of the particular applicator he is to use. *b.* No more than an erythema dose should be given without very careful consideration. *c.* The natural urge, when a procedure seems to have promise, to increase the dosage should be avoided. *d.* The method should not be made a routine procedure or a routine part of an operative procedure. *e.* Careful thought and consideration of each individual case should be given before ionizing radiation is employed."

Robbins and Schulz[30] wrote a second paper emphasizing the dangers of the present vogue of treating hypertrophied lymphoid tissue in the nasopharynx with radium.

Out of a rather large number, a few examples of unfortunate results encountered during the early years of radiation therapy at the Massachusetts General Hospital, the Massachusetts Eye and Ear Infirmary and the Collis P. Huntingdon Memorial Hospital are given to illustrate that which was discovered by the bitter experience of others in the past. Three striking photographs illustrate this statement.

In the early years the radiologist or surgeon was learning the therapeutic possibilities of ionizing radiation. In these cases late radiation necrosis had followed treatment of benign lesions, and many of the changes in normal epithelium, which were progressive and permanent, had resulted from radiation dosage that was approximately the same as the dose recommended for use in the nasopharynx for hypertrophy of lymphoid tissue. *The changes were latent, some not appearing for 10 to 20 years after treatment.*

With the idea in mind that the mistakes of the past may not be repeated in the present, certain precautionary measures are suggested: *1.* that the use of the monel applicator should not be routine; *2.* that for benign lesions in children and young people this treatment should be carried out cautiously and only after careful consideration of each case; *3.* that, except in the selected case, the treatment when given should consist of no more than erythema dose; *4.* that what constitutes an erythema dose should be determined for a given applicator; and *5.* that should no other means of determining the proper dosage for an applicator be available, the operator should estimate it by trial on his own skin.

(The unstated inference was that a dosage of irradiation dangerous for the skin would be equally dangerous to the ciliated epithelium of the nasopharynx. No reference was made to the research on this subject which had been done in Boston years before.)

Heine[11] reported, in 1936, from the Mosher Laboratory, Massachusetts Eye and Ear Infirmary, a study of the effects of radium upon ciliated epithelium. The article is clearly written and accompanied by eight excellent microphotographs. The summary and conclusions are given verbatim.

"The foregoing paper is in two parts. The first deals with some of the outstanding contributions to the literature within the last five years, pertaining to the effects of drugs upon ciliated epithelium.

"The second part has recorded observations made upon the behavior of ciliated epithelium after exposure to radiation. Three methods of approach to this problem were used; namely, the exposure of hanging drop specimens of ciliated epithelium to the action of radon seeds; second, the exposure of sections of rabbit's trachea to the direct action of Roentgen ray; third, the implantation of a fixed dosage of radium in the nostrils of rabbits and observations upon the histologic specimens made at varying lengths of time after the implantation.

"*Conclusions: 1.* Goblet cells may be another form of ciliated epithelial cells; furthermore, they are probably degenerative, as shown by changes noted in the nasal mucosa of rabbits after exposure to radium.

"*2.* The metabolic rate of the cells evidently was not affected by Roentgen ray when this metabolic rate was measured by changes in the pH of the solutions containing the tissues.

"*3.* It may be possible that some dosages of Roentgen ray are actually stimulating; therefore, there should be a dosage that will produce beneficial results.

"*4.* Ciliated epithelium is not a delicate structure. If one considers that a single dose of unfiltered Roentgen ray of eight erythemas is the maximum for the skin of man, the picture of the severity of a burn produced by a single dose three times that amount, or 24 erythemas, is almost beyond one's imagination; however, the evidence points toward the fact that these frail-appearing cells do stand such a dose with impunity.

"In searching for an answer to the question, 'Why are these cells so remarkably viable?', I can find no better explanation than the one offered by by Dr. Werner Mueller, namely, that cilia are an ancient structure biologically; that, therefore, in the order of events, they should be the last to be destroyed."

POLIOMYELITIS AND TONSILLECTOMY.

The question whether or not tonsils and adenoids should be removed from children during the months when poliomyelitis is occurring is debated in practically every hospital in the United States and Canada. Yearly articles on this problem have been reviewed here, and the problem is still unsettled.

Aycock[32] criticizes the way the question has been investigated and points out some of the defects of the various analyses in a most thought-provoking article which your editor advises should be read in its entirety.

He believes there is conclusive evidence that the virus of poliomyelitis is widely disseminated and that the paralytic disease is the exceptional manifestation of infection with the virus; and, furthermore, that there are reasons that this exceptional but serious consequence in what is ordinarily an entirely benign and trivial virus infection may be determined not so much by the circumstances of exposure to the virus but by factors which make for susceptibility in a certain few of the many who are exposed.

The articles which steadily appear voice opinions which vary all the way from the point that there is no relationship between tonsillectomy and poliomyelitis to the belief that all surgical procedures on the upper respiratory passages should be suspended during the "poliomyelitis season."

As a matter of fact, the two diametrically opposed points of view seem to take their origin not so much in any divergent evidence as in convictions stemming, on the one hand from a willingness to put any precautionary measure against poliomyelitis before other medical considerations; and, on the other hand an unwillingness to let anything interfere with convenience, if we are to believe such often heard statements as "getting ready for school" applied to the operation.

From the nature of the situation, with a very sizable proportion of the population experiencing a tonsillectomy at one time or other and only a very small fraction of the population ever suffering an attack and an even smaller fraction suffering the bulbar form of poliomyelitis, any actual association between the two would not be strikingly apparent in even a very large series of tonsillectomies, while it might easily be seen in a much smaller series of cases of poliomyelitis. In other words, one could do many tonsillectomies without encountering such a case, but one would not have to see many cases of poliomyelitis before seeing bulbar cases following the operation by a specific interval.

The evidence, furthermore, usually obtained in any statistical survey may not be at once convincing for the reason that the hazard of the disease in recently tonsillectomized individuals is numerically small, even though much larger than in the population in general. In large surveys, the excess in frequency of recent tonsillectomy in bulbar cases over that in spinal cases and the excess in the frequency of tonsillectomy over a large assortment of other "surgical procedures" when distributed over a period of 60 days amounts to a statistical dilution which makes it not at all impressive. Had these studies gone one step further to test the crucial question whether tonsillectomy preceding bulbar cases was significantly concentrated at around 14 days preceding the disease, the question of a relationship would have been settled.

There is a tendency toward continued study by broad population survey methods which, by their very nature, are not likely to afford an answer to the question which would be any more decisive (because any differences shown by them are likely to be fractional) than are the few detailed studies which are already available — or, better still, than the clinical one's imagination; however, the evidence points toward the fact that these frail-appearing cells do stand such a dose with impunity.

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As a matter of fact, the two diametrically opposed points of view seem to take their origin not so much in any divergent evidence as in convictions stemming, on the one hand from a the tonsillectomies during a visit with relatives in a distant community where poliomyelitis was present. Other cases have been presented where bulbar poliomyelitis followed closely upon tonsillectomy.

The publication of these cases resulted in a ban on tonsillectomies in most hospitals over the country during the season of the year when poliomyelitis is prevalent; namely, the Summer months. I was one of those who, as a consultant in infectious diseases, urged this ruling on the grounds that this operation is elective and could be postponed to the colder months when there was less likelihood of there being poliomyelitis virus in the nasopharynx. Pedersen and others have given us some interesting food for thought on this matter. Last year I revised my opinion on this, recommending that the ideal time for tonsillectomies was immediately after the close of school in June in this part of the country; also, that tonsillectomies could continue through the Summer months providing there were no cases of poliomyelitis being reported from the community and no known possibility of exposure from outside. In Massachusetts last Summer the state board of health through weekly bulletins kept hospitals informed of the situation of poliomyelitis in this state, as well as over the country at large. It would seem that this method offers a satisfactory solution to the tonsillectomy problem; nevertheless, there is always the warning of the "K" family, and regardless of season, you who take the responsibility of performing an elective operation should be aware of the fact that possible exposure at other seasons may occur. In this discussion we have come to realize that other dangers besides poliomyelitis confront this operation, and that to be forced to carry it out when upper respiratory infections are most prevalent is a disadvantage to be seriously considered. That we were carried too far in our zeal to prevent poliomyelitis through a seasonal ban on tonsillectomies is now clear, but this is just one of many errors that have been made in the numerous efforts in fighting this disease. Until we have some quick and practical method of determining the presence of the virus of poliomyelitis in patients on whom tonsillectomy is contemplated, we must rely upon epidemiological data as supplied in Massachusetts.

Cunning[34] summarizes data provided for 1948 on tonsillectomy and poliomyelitis in 10 tables which cannot be given here, and deserve study. After 10 years' study on this subject, following over 17,000 poliomyelitis cases and 35,000 tonsillectomy cases, he is still of the same opinion as he was one year ago, namely, that no definite causal relationship between tonsillectomy and poliomyelitis has been established; therefore, he does not believe that tonsillectomy should be postponed indefinitely simply because the Summer months are the months during which poliomyelitis is prevalent. If, however, there is a distinct rise in the poliomyelitis rate, bordering on epidemic proportions, in any community, all elective operations should be postponed.

Until evidence of a conclusive nature is forthcoming, the surgeon should decide whether or not to perform tonsillectomy during the Summer months and whether the dangers of delaying the operation are greater than the still undetermined risk of increasing susceptibility to poliomyelitis.

The Journal of the American Medical Association wrote an editorial[35] commenting on Cunning's report which has been summarized above. The editorial is condensed as follows:

The journal (March 21, 1942) warned that poliomyelitis, particularly the bulbar form, greatly increased in children after tonsillectomy or adenoidectomy in epidemic areas. . . . Since that time avoidance of such operations during epidemics has been almost universally adopted. Recently, otolaryngologists seem inclined to the belief that a causal relationship does not exist between the two and that bulbar poliomyelitis following tonsillectomy is probably purely coincidental. Cunning as a result of a survey of data covering 1947 felt that the operation should not be indefinitely postponed simply because Summer months are the months that poliomyelitis is prevalent. This report was recently reviewed by Faber (given later), who concluded that proofs previously offered for a causal relationship between incidence of poliomyelitis and tonsillectomy during epidemic times were still valid and that evidence for this had not been refuted by the Cunning survey.

The weight of past and recent evidence favors the existence of such a relationship. As tonsillectomy is an elective operation, which rarely requires immediate surgical attention, the preventive attitude of delay is justified. Until conclusive evidence is presented that such procedures do not increase incidence of poliomyelitis the inadvisability of operation during periods of epidemic poliomyelitis should be emphasized.

Cunning[34] replied to the above editorial, making a correction and a pertinent criticism. He states the editorial failed to mention the 16,643 cases of tonsillectomy performed and carefully followed in 15 different states throughout the country for a period of two months after operation, in none of which did bulbar poliomyelitis develop.

For four years the Committee for the Study of Poliomyelitis and Tonsillectomies of the American Laryngological, Rhinological and Otological Society, Inc., has honestly attempted to learn the truth concerning the relationship between poliomyelitis and tonsillectomies. This committee, a large number of the country's leading epidemiologists and many local health authories, agrees on two main points: *1.* tonsillectomies and adenoidectomies or any other elective operation should not be performed in a community where poliomyelitis (or any other contagious disease) assumes epidemic proportions, and *2.* tonsillectomies and adenoidectomies can with safety be performed in a community where poliomyelitis (or any other contagious disease) does not assume epidemic proportions.

The effort is being made yearly by statistical study to discover the truth regarding any possible relationship between tonsillectomy and poliomyelitis. One of these is in a sense the official report of the American Laryngological, Rhinological and Otological Society, Inc., and a summary of such report has been made each year in this place. Statistics are, however, unsatisfactory masters and may be read in several ways. The report summarized last year received the following criticism by Faber.[37]

In a recent publication, Dr. D. S. Cunning presented the results of a survey conducted by a committee of the American Laryngological, Rhinological and Otological Society, Inc., on poliomyelitis and tonsillectomy for the year 1947 and also described the results of a similar survey for 1946. He summed up his conclusions as follows: "I am more than ever convinced that there is no causal relationship between the two, and that when a bulbar poliomyelitis follows a tonsillectomy it is coincidental."

This dangerous conclusion, which I fear is already being widely accepted by otolaryngologists, should be very carefully weighed in the light of the evidence presented by the present report and of other evidence totally disregarded by it.

As regards the Cunning data, the following points deserve consideration:

1. Twenty-four cases of poliomyelitis in the reported series actually did follow tonsillectomy in 1947 and 62 cases in 1946. Of these, six, or 25 per cent, were bulbar in 1947, and 18, or 29 per cent, in 1946, whereas the incidence of bulbar poliomyelitis in all cases of poliomyelitis covered by the survey was 11.6 per cent in 1947 and 15.4 per cent in 1946; that is, the incidence of bulbar forms was slightly more than twice as high in persons for whom tonsillectomy has been done as in those for whom it has not.

2. Since the compilation by Aycock, in 1942, and the editorial of March 21, 1942, in the journal tonsillectomy has come to be generally avoided during epidemics, and to a smaller extent even during the Summer and Fall months, because of the widespread fear of poliomyelitis that is felt both by physicians and laity; the result of such avoidance would, of course, be to reduce greatly the incidence of post-tonsillectomy cases and thus to increase the proportion of non-post-tonsillectomy cases.

3. The proof of a relationship between tonsillectomy and poliomyelitis is positive in respect to the high proportion of bulbar cases and cannot be blindly disregarded. Thus Aycock's summary shows 170 cases in which poliomyelitis followed tonsillectomy within 30 days (approximately the upper limit of incubation), of which 121, or 71 per cent, were bulbar, whereas in the 30 to 60-day period only 20 per cent were bulbar. The Cunning survey used a 60-day interval, which may explain the lower incidence of bulbar poliomyelitis than in the 30-day reports. The extraordinary report by Krill and Toomey deserves mention. Five children in one family were subjected to tonsillectomy on the same day, and all came down with bulbar poliomyelitis 10 to 12 days later, three of them dying of the disease. Since the Aycock compilation, numerous other reports of post-tonsillectomy poliomyelitis have appeared (including the 86 of the two Cunning reports) and the 17 of Anderson's paper. The latter particularly should not be disregarded, since it offers clear proofs of the relationship under discussion. Anderson obtained data on tonsillectomies performed in Utah in 1948, both before and during the heavy epidemic of that year. Before (July) the outbreak, the incidence of poliomyelitis after tonsillectomy was 0.7 per thousand; in August, when the epidemic began, it was 1.4 per thousand and at the height of the epidemic it was 19.2 per thousand tonsillectomies (these figures have been calculated from the original data). All the post-tonsillectomy cases of poliomyelitis were bulbar. The incidence of poliomyelitis in children in whom tonsillectomy had recently been done was 2.6 times, and of bulbar poliomyelitis 16 times greater than in the general child population.

The Cunning survey is, therefore, in my opinion, a dangerous one, particularly since it is in a sense the official report of the American Laryngological, Rhinological and Otological Society, Inc., and is apt to obtain wide acceptance, at least among specialists in that field, and might lead to the abandonment of what is now generally accepted as an important means of prevention of poliomyelitis. That the author himself still entertains doubts of the correctness of his views is indicated by a remark that follows the conclusion already quoted: "I do not advocate the indiscriminate removal of tonsils during any severe epidemic and have no desire to place our children's lives in jeopardy through injudicious surgery...." It is to be hoped that this sentiment will continue to be felt by all physicians who are responsible for deciding on tonsillectomy, especially during epidemics of poliomyelitis.

SURGERY.

Stoner and Freeman[38] believe the rhinologist owes it to the obstetrician, the pediatrician and the general practitioners to emphasize properly the seriousness of congenital posterior choanal occlusion and to recognize it as a possible cause of the symptoms in cases of immediate difficulty in nursing or breathing of the newborn. They describe such a case and illustrate their treatment, using the inner cannula of a No. 2 tracheotomy tube in each side of the nose to keep the surgically made openings patent. The obturators were removed on the seventeenth postoperative day and at the ninth postoperative month both posterior choanal openings were patent.

Sheehan and Swanker[39] write a short, abrupt résumé of the methods of surgically attacking congenital choanal atresia and conclude that while slightly more difficult in some respects, the transpalatine route affords the most satisfactory surgical approach. The operation they advocate is a combination of Ruddy's incision, Neto's fenestration and Blair's treatment of the nasal mucosa and is outlined in nine steps.

Hanckel[40] reviews various methods of dealing with congenital bilateral choanal atresia and describes in detail and with many illustrations a case which required many interferences over a period of four months to cure. Other authors have likewise advocated a transpalatine approach and have suggested that uneventful quick recovery is the rule. This author is to be admired for writing so plainly about the troubles that may be encountered and his paper is recommended to every otolaryngologist who has to deal with this condition.

Dolowitz and Holley[41] review the literature and find that surgical repair of congenital choanal atresia has, in the past, rarely been successful. Failure has almost always been due to secondary scar formation which closes the opening, despite attempts to produce a permanent fistula by means of skin grafts, prostheses or repeated cauterizations. Even in those instances in which a permanent fistula has been made there has usually not been a return to normal nasal function owing to the fact that no ciliated epithelium lined the new opening.

They describe a new approach to this problem which permits the obstructing bone to be removed by a transpalatine technique and a new nasal passage to be formed which is lined with flaps of ciliated mucous membrane. Seven drawings adequately illustrate the procedure.

One case is reported in which this technique was used. The patient, an eight-year-old child with unilateral congenital choanal atresia, was discharged from hospital eight days after admission and five days after operation. She has experienced entire relief of her nasal complaints since the operation 31 months ago. The emotional and social readjustment following the alleviation of this minor defect was striking.

Shapiro[12] states that in eight of 12 reported cases of cysts on the base of the tongue in infants the diagnosis was made at postmortem examination. In three of the four cases in which the diagnosis was made in a living child simple incision and drainage resulted in complete disappearance of the cyst. In the fourth case, which was really in the body of the tongue, complete incision via the undersurface of the tongue resulted in cure.

The symptoms usually date back to the very earliest infancy and characteristically vary in the degree of severity from time to time. These infants have a peculiar cry and inspiratory stridor. Intermittently they have attacks of complete respiratory obstruction with cyanosis. Death may occur in one of these attacks. Posturing of the patient may give partial relief from the respiratory difficulties.

He reports a case, pictures a postmortem specimen and a diagram of the conditions existing in a case of a cyst at the base of the tongue.

By direct laryngoscopy the diagnosis should be simple. A carefully used tongue depressor or simple palpation may reveal the presence of the cyst. (The finger may be too large for palpation, the tongue depressor and the laryngoscope may hide the mass. Tongue forceps should be used to pull the tongue forward and the suspected area seen with the use of a head mirror.)

NEW GROWTHS.

Grotts[13] describes minutely a transitional cell carcinoma of the nasopharynx in a two-year-old child. Five weeks before admission a retropharyngeal abscess was diagnosed and drained. She was given three weeks of penicillin therapy but did not improve and lost weight. The posterior nares were completely obstructed with thick mucus; the soft palate was pushed downward and forward by a rather firm mass which protruded into view posteriorly and appeared dark brown, crusted and necrotic. There was marked surrounding edema and cellulitis. Biopsy resulted in the diagnosis of a transitional cell carcinoma of the pharynx. Following this diagnosis 11 deep X-ray treatments of 210 r. each were given to both the left and the right nasopharynx. She died at home 10 weeks after the onset of symptoms. No necropsy was obtained.

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THE PROGRESSION OF IMPAIRED HEARING FOR HIGH TONES DURING CHILDHOOD.*

STACY R. GUILD Ph.D.,
Baltimore, Md.

INTRODUCTION.

Impaired hearing for high tones only, of the type termed "abrupt" high-tone loss in Guild's method of classifying audiograms (1932), occurs both in adults and in children. Of the 1,700 persons 20 to 69 years included in Ciocco's (1932) study of the hearing of 1,980 individuals, who were randomly selected and whose auditory thresholds for pure tones were detemined audiometrically over the range of frequencies from 32 to 16,384 cycles per second, inclusive, 794 persons had some form of a simple "abrupt" type of loss for high tones, and an additional 231 persons had this type of hearing loss complicated by the presence of a "dip" at some frequency lower than that of the "drop." In other words, Ciocco's report indicates that about three-fifths of all adults have some form or other of an "abrupt" type of impaired hearing for high tones.

The incidence of the "abrupt" type of high-tone loss is much lower in children than in adults. The largest randomly selected group of children that can be used for comparison with Ciocco's data on adults is the group of 1,365 Baltimore school children, aged eight to 13 years, reported by Guild, *et al.*, in 1940.[10] The hearing acuity of these children was tested for the same tonal frequencies as were the adults of Ciocco's study, and in both studies the same method of classification of the audiograms was used. Slightly over 15 per cent of the audiograms of the first hearing tests made of the ears of this group of 1,365 children belong to one or another of the sub-categories of the "abrupt" type of high-tone loss. From these two studies, the incidence of the "abrupt" type

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*From the Johns Hopkins University School of Medicine.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 13, 1950.

of hearing impairment for high tones appears to be about four times as great in adults as in children.

CAUSE OF "ABRUPT" TYPE HIGH-TONE LOSS.

In adults whose ears have been studied histologically, an "abrupt" type of hearing impairment for high tones, at least when the loss of normal acuity begins at the frequencies of 8,192, 5,793, 4,096, 2,896 or 2,048 cycles per second, is usually associated with a more or less extensive absence of the organ of Corti in the basal turn of the cochlea.[5] No histologic observations are available on the ears of children with this type of impaired hearing. The idea that the immediate cause of "abrupt" high-tone loss is the same in children as in adults; namely, an absence of the organ of Corti in part of the basal turn of the cochlea, is strengthened by Guild's recent report[9] that in 52 cases of "abrupt" high-tone loss in children, who were re-examined in each case six years or more after the first test had been made, there was not even one instance in which the highest tone that was well heard was of a higher frequency at the last examination than it was at the first examination. The hearing of some ears with this type of loss had become worse, none had improved.

The remote, or true, cause of impaired hearing for high tones, of the "abrupt" type of loss, can be determined with certainty in only a small proportion of the large number of cases that are encountered by otologists and by others interested in hearing. Sometimes there is a definite history of exposure to acoustic trauma of the blast, or detonation, type so severe that the patient's ears "rang" for days or weeks afterward. In such cases, the diagnosis as to the remote cause of the impairment can be made with a high degree of confidence that it is correct, because there is not only good clinical evidence that exposure to the blast type of acoustic trauma often is followed by an "abrupt" type of high-tone loss but also good experimental evidence that the exposure of animal ears to detonation waves causes a histologically demonstrable destruction of the organ of Corti.[6,11,12,13]

When a cleancut history of exposure to acoustic trauma of the blast type cannot be elicited, even by prodding the patient's memory, decision as to the remote, or true, cause of "abrupt" type high-tone loss is usually uncertain. Most commonly, in such cases, especially when the patient is a child, the hearing impairment is classified as "congenital" and ascribed either to a failure of the basal turn of the cochlea to develop normally or to a toxic damage of this part of the organ of Corti, or of the nerve fibres from it, by some medication received either by the mother before delivery or by the child during early infancy. Naturally it is difficult to prove, or to disprove, the correctness of such a diagnosis; at best it is unsatisfactory to the otologist who makes it, if not to the patient or to his parents. Many otologists frankly recognize such diagnoses as belonging to the "I don't know" category.

The only satisfaction a conscientious otologist has in making such a speculative diagnosis is the certainty that, whatever the true cause of the impaired hearing may have been, he is not neglecting his duty to his patient when he tells him that nothing can be done to restore the hearing for the tonal range affected by the lesion present.

Fortunately for the afflicted persons, hearing for the conversational voice, either spoken or recorded, is seldom seriously affected by an "abrupt" type of hearing impairment for high tones, except when the impairment is of extreme degree (sub-categories C_8 to C_{12} of Guild's classification of audiograms.)[7] When the loss begins at the frequency of 4,096 or of 5,793 cycles, or yet higher, the patient seldom realizes, until his hearing is tested with a pure-tone audiometer, that he has any hearing defect whatever.

Two reasons are responsible for the fact that almost nothing is known about the rate of development or of progression of impaired hearing for high tones, of the "abrupt" type, except in the cases definitely caused by exposure to acoustic trauma. In the first place, because patients with this type of hearing impairment are unaware of the condition, they cannot usually even offer subjective impressions with respect to the time of onset or the rate of progression of the inability to hear very high pitched tones. In the second place, because otologists have had nothing effective in the form of treatment to offer such patients, they have seldom urged them to return periodically, even for observation, and when they have done so, the patients have seldom come, due to lack of interest. The result is a paucity of follow-up observations on persons who have an "abrupt" type of high-tone loss.

OBSERVATIONS.

The staff of the Otological Research Laboratory at the Johns Hopkins University, aware of the above fact, has in past years encouraged individuals with an "abrupt" type of high-tone loss, whatever the supposed cause, to return from time to time for re-examination, free of any charge or obligation. As elsewhere, not many such patients have actually returned over a period of years. So far as concerns adults, the observations that have been made are in general agreement with the impressions that have also been gained elsewhere (as evidenced in personal conversations of the writer with otologists from this country and from abroad), namely, that impaired hearing of any of the "abrupt" high-tone loss sub-categories remains stationary unless or until there is superimposed on it the effect of some other lesion that causes deafness, a lesion essentially different from the cochlear one supposedly responsible for the high-tone loss present at the first examination, or until additional exposure to severe acoustic trauma causes an extension of the cochlear lesion. The writer hopes in the near future to make a separate report of the observations that have been accumulated with respect to the progression of impaired hearing for high tones in adults; the present report will, except for the above general statements, be limited to a consideration of some interesting observations that have been made with respect to the development and the progression of "abrupt" type high-tone losses in children.

The opportunity to observe the development and the progression of "abrupt" type high-tone losses in children came not as a result of the direct efforts to follow patients with this type of hearing loss but as a by-product of the departmental program to prevent deafness in children by irradiation of nasopharyngeal lymphoid tissue. This program has been carried on in the department for many years, and intensively since 1939. A large number of children have had the irradiation treatment; some of them were participants in a special study made of the hearing of school children,[10] some were patients in the Out-Patient Department of the Johns Hopkins Hospital, and some were private patients of members of the clinical staff of the Sub-Department of Otolaryngology. An essential part of the program has of course been the follow-up examinations. To date, the most comprehensive report of the follow-up study, insofar as the effect of nasopharyngeal irradiation on hearing acuity is concerned, is that made recently by Guild,[9] one item of which has already been mentioned as confirmatory of the idea that the immediate cause of "abrupt" high-tone loss in children is the same as has been proven by histologic observation to be the usual cause in adults.

The present report of this follow-up program concerns children who, while under observation, either developed an "abrupt" type of high-tone loss gradually or had an already existing impairment of this type progress markedly. The great majority of the children with impaired hearing for high tones only have not, when examined several years later, had much progression of the impairments originally present. If marked progressions of impairments for high tones were of common occurrence during the childhood years, the average increase in the high-tone losses for the group of children included in the previous report[9] would have been much greater than actually occurred during the more than six years of observation.

All of the hearing tests included in the present report, like those on which the previous one was based, were made in a sound-proof room at the Otological Research Laboratory, by experienced, well-trained examiners. At each test session, a Western Electric Company 1-A audiometer was used to determine the air-conduction thresholds for tones of 14 frequencies (32, 64, 128, 256, 512, 1,024, 2,048, 2,896, 4,096, 5,793, 8,192, 10,321, 13,004 and 16,384 cycles per second), a 512 d.v. steel tuning fork was used for Weber, Schwabach and Rinne tests, and the hearing acuity for the voice was tested with a W. E. Co. 4-A phonographic audiometer, using the two-digit records. The opposite ear was suitably masked, routinely, during the making of the Schwabach, the Rinne and the voice tests, also, when indicated, during the testing with the pure-tone audiometer. The masking noise was generated in a headphone by a calibrated masking device, locally built,[2] that has been used with satisfaction in the laboratory for many years.

An example of an impairment that did not progress significantly during a nearly 10-year period of observation is included in the case reports given below (see Case 5); comparison of this record with those of the other cases illustrates the difficulty of predicting which children are going to have progression of their impairments during any given period of time.

The four cases of progression or of development of an "abrupt" type high-tone loss reported below include the two most striking examples (see Cases 1 and 3) that have been observed; the selection otherwise has been on the basis of cases that illustrate different forms of progression.

Cases more or less similar to the ones here reported have doubtless been observed by other investigators, but probably, by any one investigator, in such small numbers that publication of the observations did not seem justified. It is hoped that the present report will stimulate others to report the cases they have seen and that from the total assemblage of records better conclusions, especially with respect to causation and to prognosis, can be drawn than are warranted from consideration of the material now available to any one of those who are at present interested in the topic of "abrupt" type high-tone loss. To facilitate the use of these records by others, for the purposes indicated above, the clinical facts about each of the cases here reported are given in more detail than would otherwise seem warranted.

CASE REPORTS.

Case 1: This white boy, J. A., was 10 years old at the time the first examinations were made as part of the study of Baltimore school children.[10] Previously, from the age of 2 years to 5 years, he was seen several times in the Ear, Nose and Throat Dispensary of the Out-Patient Department of the Johns Hopkins Hospital, because of recurring attacks of bilateral suppurative otitis media and cervical adenitis. In 1934, at the age of 5 years, tonsillectomy and adenoidectomy was advised, but the parents refused permission to operate. Three years later, at the age of 8 years, after more episodes of otitis media, the advice to have his tonsils and adenoids surgically removed was accepted and the operation was performed in 1937. Hearing tests were not made at this time.

The physical examination made at the time of the first hearing tests (Dec. 19, 1939) showed that a regrowth of adenoids had occurred; the tubal orifice on the right side was so completely overgrown by lymphoid tissue that it could not be seen through a nasopharyngoscope, on the left side the tubal orifice was partially overgrown by lymphoid tissue. Tonsillar tags were present in both fossae, and the walls of the oropharynx had hyperplastic lymphoid tissue. He reported having had only an average number of colds (2 or 3 a year each of which lasted about a week) and said he had had no recurrence of otitis media since his operation. Both tympanic membranes were scarred, the left one in the pars flaccida as well as in the anterior-inferior quadrant of the pars tensa, and both were moderately thickened and moderately retracted. Nasal breathing space was good, and the sinuses transilluminated clearly.

The boy said his hearing was good, both ears, as indeed it was except for the tones with frequencies above 4,096 cycles (see audiograms of Dec. 19, 1939, at top of Fig. 1). In the phonographic audiometer test he correctly repeated all the numbers. The sound of the 512 d.v. steel tuning fork was not lateralized from midline positions of the head, this fork was heard better by air conduction than by bone conduction with each ear, and the bone-conduction time for each ear, with the other one suitably masked, was normal for the fork used.[2]

Irradiation of the nasopharynx was advised because of the combination of overgrowth of the tubal orifices by lymphoid tissue, retracted tympanic membranes and impaired hearing for high tones. The advice was accepted and treatments were given, by the use of radon in a brass applicator with walls 1 mm. in thickness, on Jan. 9, Feb. 20, March 27, May 7 and Nov. 14, 1940. All the treatments were given by Dr. Crowe personally.

The examinations made on May 7, before the treatment of this date was given, showed the nasopharyngeal lymphoid tissue to have shrunk until only a moderate amount remained; the left tubal orifice was now open but was somewhat edematous in appearance, and the right tubal orifice was only partially overgrown instead of being completely covered by lymphoid tissue as it had been before treatment was begun. The only other change revealed by the physical examination was that the degree of retraction of the pars tensa of the left tympanic membrane had increased from a rating of moderate to that of marked. In spite of the favorable changes in the nasopharynx, the hearing impairment for high tones had increased, particularly on the left side (compare audiograms of Dec. 19, 1939, and May 7, 1940, in Fig. 1). No change had occurred in his ability to hear the voice (phonographic test) or in the responses to the fork tests.

This boy was next examined after the Summer season, on Oct. 15, 1940, at which time both tubal orifices were normal in appearance, but the right as well as the left tympanic membrane now showed a marked degree of retraction and the hearing impairment for high tones had further increased for both ears (the audiograms for this date are not reproduced in the figure). He was not given another treatment on this date, but when seen a month later he was given his fifth, and final, irradiation treatment, after the hearing tests had revealed a yet further increase in the impairment of thresholds for high tones. By this time the impairment for the left ear, which had been definitely the better in all the previous tests, was about as great as for the right ear (audiograms of Nov. 14, 1940, Fig. 1), which also had suffered a progressive loss of acuity for high tones during the first year of observation.

During the next year this boy was examined on three occasions but was not given further treatment; the lymphoid tissue in his nasopharynx continued to shrink until only a very small amount remained. Both tubal orifices were graded as entirely normal in appearance throughout the year, and the degree of retraction of the tympanic membranes decreased to a rating of moderate. By the end of the year the threshold for the very high frequency of 13,004 cycles had markedly improved in both ears (see audiograms for Nov. 4, 1941, Fig. 1), but the threshold for 8,192 cycles had become worse in both ears, and in the left ear the threshold for 5,793 cycles was poorer than in the right ear, which previously had been much the worse for this tone. The results of the voice and of the fork tests remained unchanged.

The boy was examined again on March 14, 1942, at which time all the findings, including those with respect to hearing acuity, were essentially as they were in November, 1941. He was not seen again until 5 years later, after the war, when he returned, at request, on April 5, 1947. He was then 17 years of age and was working in dusty surroundings, which may account for his statement that he had had many "colds" the past year. Otherwise he had no complaints referable to his nose, throat or ears. The tonsillar tags were still present, but as previously seemed to be causing no trouble. The amount of lymphoid tissue in the oropharynx had decreased greatly, and the nasopharynx was free of lymphoid tissue except for a tiny amount in each fossa of Rosenmueller. Both tubal orifices were normal in appearance. The examiner on this occasion graded the degree of retraction of the right tympanic membrane as moderate and that of the left one as marked. The thresholds for the two ears were now practically identical for high tones (audiograms of April 5, 1947, Fig. 1); the improvement for 13,004 cycles had been lost and the audiograms were typical examples of the "abrupt" high-tone loss type, with 5,793 cycles the lowest frequency involved.

It is interesting to note that during the somewhat more than seven years of observation the threshold for the "border" tone, of the frequency of 5,793 cycles, dropped 35 decibels in the right ear (from the 30 to the 65 db. level) and 55 decibels in the left ear (from the 10 to the 65 db. level.

Comments, especially with respect to causation of the impairment:

The remote cause of the hearing impairment for high tones that developed in this boy is unknown. The immediate cause,

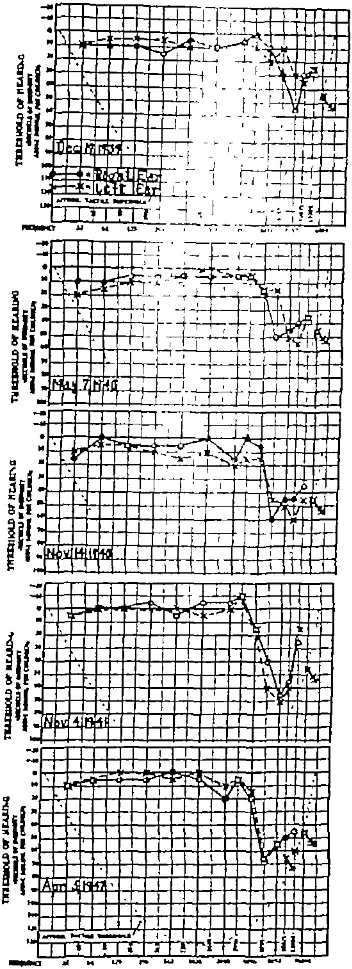

Fig. 1. Five of the 11 pairs of audiograms that were made of a boy who was 10 years old when the first tests of hearing acuity were made and 17 years old when last examined. These audiograms show the progressive increase in the impairment of thresholds for high tones that occurred gradually, and bilaterally, during the period of observation. See text, Case 1.

for each ear, was probably a progressive atrophy of the organ of Corti and of the external sulcus cells in the lower 7 mm. or so of the basal turn of the cochlea, and a partial atrophy of the nerve that supplies this region of the auditory end-organ.[5] There is no reason to believe that the treatments this boy received, either the surgical removal of tonsils and adenoids previous to the making of the recorded hearing tests or the nasopharyngeal irradiations administered during the first year of the observed progression of his hearing impairment for high tones, were in any way responsible for the hearing loss that developed. It also seems most improbable that this boy's recurrent attacks of suppurative otitis media up to the age of 8 years in any way caused his eventual hearing loss for high tones; many audiograms similar to his on April 5, 1947, shown in Figure 1, have been recorded for persons whose histories with respect to otitis media are negative and whose tympanic membranes are normal in appearance.

Furthermore, the gradual course of the development of the functional impairment of this boy's ears practically rules out of consideration as the cause any acute systemic disease, such as mumps or any other of the viral or bacterial infections. It is difficult to imagine localized lesions due to infections, or to toxic agents of any kind, that progress for years after the systemic symptoms have disappeared and that eventually cause bilaterally symmetrical impairments of hearing for high tones.

Had this boy's hearing first been examined when he was 17 years old, after his impairments had reached the stage shown in the audiograms of April 5, 1947 (see Fig. 1), neither acoustic trauma, in the form of exposure to a detonation, nor a congenital failure of the basal turn of both cochleae to develop normally, could have been ruled out as explanations for the symmetrical functional defects present. The boy thought he had good hearing, he had never noticed any change in keenness of hearing except during the attacks of otitis media, and otherwise at the age of 17 years he would have given a history typical of those obtained from persons whose impairments are commonly regarded as due to congenital cochlear defects or to an entirely forgotten episode of exposure to an explosion. As it is, both of these possible causes of this type of hearing impairment can definitely be eliminated from consideration.

Whether or not this person's hearing impairment for high tones will progress beyond the present stage (as shown in the lowest pair of audiograms in Figure 1) before either normal ageing or other factors deleterious to hearing become superimposed on the existing lesions, is a matter that can be answered only by the passage of time and the making of further careful examinations of his hearing ability. I personally believe that within the next decade of his life the ability to hear the tones for which the thresholds are already severely impaired will be completely lost, but that unless this man is exposed to severe acoustic trauma or has another episode of otitic infection or suffers some less common cause of deafness, he will continue to have normal hearing for speech until presbyacusic changes affect his understanding of consonants. This optimistic opinion is based on the absence, in my experience, of observations in adults of "spontaneous" changes in hearing for high tones comparable to those that occurred during childhood in this boy.

Case 2: This white boy, J. S., was 8 years old when first examined in the Ear, Nose and Throat Dispensary of the Out-Patient Department of the Johns Hopkins Hospital. In spite of a history of frequent attacks of bilateral otitis media and many myringotomies, his tympanic membranes were both normal in appearance except for a marked retraction of the pars flaccida, and the hearing was remarkably good except for the extremely high-pitched tones (see Fig. 2, audiograms of Oct. 10, 1939). He had large tonsils and adenoids, and both tubal orifices appeared to the examiner to be partially occluded by lymphoid tissue. Tonsillectomy and adenoidectomy were performed, at the Johns Hopkins Hospital, Dec. 14, 1939. Hearing tests were not made when he returned a month later for his post-operative check-up, but when he came back 3 months later the audiograms revealed that a marked increase had occurred in the impairment for high tones in the right ear and a slight increase in the left ear (see Fig. 2, audiograms of April 4, 1940). The tonsillar fossae were clean; a moderate amount of lymphoid tissue was seen in the nasopharynx but both tubal orifices were clear. The tympanic membranes now had a moderate degree of retraction of the pars tensa. On April 15, 1940, his nasopharynx was irradiated by Dr. Crowe (2.2 gram-minutes equivalent to each side, using the radon applicator with a brass wall 1 mm. thick). Three weeks later (see audiograms of May 8, 1940, Fig. 2) hearing acuity with the left ear was impaired for all tones, and the "abrupt" type high-tone loss of the right ear had increased slightly for the frequency of 4,096 cycles. The left ear had an 18 per cent loss for voice (phonographic audiometer test), and the sound of the 512 d.v. steel tuning fork, which had previously not been lateralized from midline points of the head, was referred to the left ear, for which ear the sound was louder by bone conduction than by air conduction and the bone-conduction time was normal. Otoscopic examination of the left ear revealed only a slight hyperemia of Shrapnell's membrane and a moderate retraction of all parts of the tympanic membrane. The boy had a "cold," which had begun about a week previously; no treatment was given this day.

When he returned at the end of the Summer season the hearing of the left ear had improved, but the right ear showed a 40 decibel increase in the previous impairment for 4,096 cycles (audiograms of Sept. 24, 1940, Fig. 2). Both tubal orifices appeared, on nasopharyngoscopic examination, to be partially occluded by lymphoid tissue, and another irradiation treatment was given this day by Dr. Crowe (2.5 gram-minute equivalent, to each side, with the 1 mm. thick brass-walled applicator). A third treatment of 2.44 gram-minutes, with the same applicator, was given Dec. 2, 1940, at which time the hearing was essentially the same as it was in September (audiograms not reproduced). He was examined again in May, 1941, and in October, 1943, but was not given further treatment. He returned, at the request of the Otological Research Laboratory, on April 3, 1947. He reported that he had only occasional mild colds and no sore throats. The tonsillar fossae were clean; a small amount of lymphoid tissue was present in the nasopharynx but both tubal orifices were clearly visualized on nasopharyngoscopic examination. The examiner on this occasion (Dr. Walter Loch) recorded both tympanic membranes as markedly retracted, pars tensa as well as pars flaccida, and as showing definite scarring. The hearing acuity, about 7½ years after the first tests, is shown in the lowest part of Figure 2.

Comment: The threshold for the tone with a frequency of 8,192 cycles was the same in the last test of the left ear as it was in the first test, and for the frequencies adjacent to 8,192 cycles (5,793 and 10,321) in the series tested the thresholds had changed but little. The thresholds for all the other frequencies above 1,024 cycles were, however, definitely poorer for the left ear at the last test than at the first; the greatest change was for 4,096 cycles, at which frequency the audiogram of the left ear has a marked "dip." Whether or not the hearing loss for the left ear of this boy will progress until it reaches the condition present in the right ear is unknown; personally I think the pathologic process in the left ear, whatever it is, had not yet become stabilized when the boy was last examined.

It is of interest to note that for the right ear of this boy essentially all the impairment that developed during the period of observation, except for the "border" frequency of 4,096 cycles, occurred before the nasopharyngeal irradiation treat-

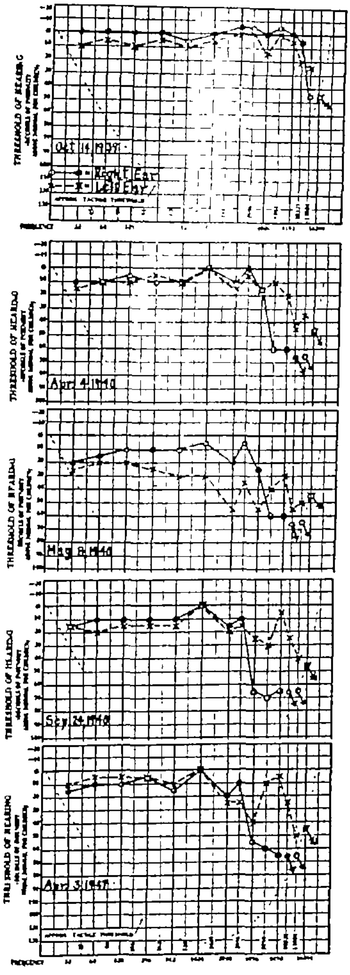

Fig. 2. These 5 pairs of audiograms, of the 8 made, show changes in hearing acuity that occurred during 7½ years in a boy who was 8 years old when the first hearing test was made. See text. Case 2.

ments were started. No hearing tests were made when he was admitted to the hospital for operation, therefore it is impossible to date the loss with respect to the tonsillectomy and adenoidectomy, which occurred 2 months after the first audiograms shown in Figure 2 were made. As with Case 1, I doubt that either the surgical or the irradiative treatment of this boy was related, except in point of time of occurrence, to the progression of his hearing impairment for high tones.

The behavior of the "border" tone of the loss in the right ear of this boy, 4,096 cycles, was similar to that of the "border" tone, 5,793 cycles, of the left ear of Case 1. In both instances the gradual progression of the impairment for the "border" tone shifted the edge of the "abrupt" type high-tone loss first present (audiogram of May 7, 1940, for the left ear of Case 1; audiogram of April 4, 1940, for right ear of Case 2) downward half an octave in pitch.

Case 3: N. B., a white boy, was 12 years old when first examined, on Nov. 9, 1939, as a participant in the study of the hearing of Baltimore school children.[10] The history he gave was entirely negative for ear, nose or throat troubles, except for colds, of which he had about three a year. Nasal breathing space was good; the sinuses transilluminated clearly. The tonsils were small, but the adenoids were large and partially covered both tubal orifices. The tympanic membranes were not thickened or scarred but appeared to be moderately retracted in all parts. With the phonographic audiometer test he correctly repeated all the numbers, and the results of the tests with a 512 d.v. steel tuning fork were likewise entirely normal. The only departures from normal hearing acuity that were found were the impairments for very high tones, shown in the audiograms of Nov. 9, 1939, of Figure 3.

On the basis of the combination of overgrowth of the tubal orifices by lymphoid tissue, retraction of the tympanic membranes and impairment of thresholds for some of the high tones, nasopharyngeal irradiation was advised by Dr. Crowe. The advice was accepted by the boy's parents, and three treatments were given (Nov. 28, 1939; Jan. 4, 1940; April 3, 1940) with radon in a brass applicator that had a wall thickness of 1 mm. The tests made on the day of his second treatment showed that his hearing impairment for high tones had increased considerably; this increased impairment was confirmed by tests made in February and in April. The stages of impairment that had been reached by May 6, 1940, a month after his last irradiation treatment, are shown in the second pair of audiograms reproduced in Figure 3. By this time only a small amount of lymphoid tissue remained in the nasopharynx, both tubal orifices were normal in appearance on inspection with a nasopharyngoscope, and the degree of retraction of both tympanic membranes had receded to a rating of slight.

The appearance of the nasopharynx and of the tympanic membranes remained essentially unchanged during the next 8 years, but the hearing

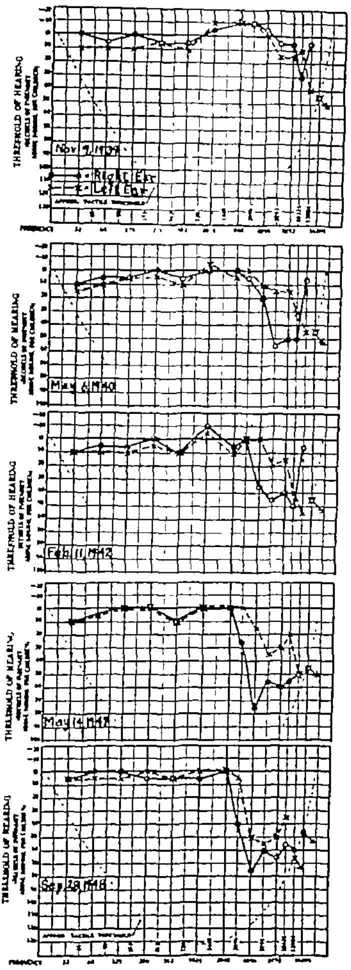

Fig. 3. Five of the 12 pairs of audiograms that were made during a nearly 9-year period of observation of a boy who was 13 years old when first examined. The marked degree of bilaterally impaired hearing for high tones developed gradually. See text, Case 3.

impairments for high tones progressed, in the manner shown in the audiograms reproduced in the lower three parts of Figure 3.

Comments: The 5 pairs of audiograms reproduced in Figure 3, of the 12 tests made during the nearly nine-year period of observation, suffice to show not only the amount of the increase in the impairments of thresholds for high tones that occurred bilaterally but also the difference in the order of progression in the two ears of this boy. In the right ear the progression occurred as a deepening and widening, towards lower tones, of the moderate "dip" for 10,321 cycles that was present when he was first examined (audiogram for right ear on Nov. 9, 1939). Only after more than 2 years of this type of progression did the threshold for 13,004 cycles, at the high-frequency side of the "dip," begin to be impaired. During its drop from the normal threshold of 5 db. to that of 60 db., the threshold for the "border" tone at the other side of the widened "dip" also dropped from a normal of "zero" to the 40 db. level (2,896 cycles, right ear, audiograms of Feb. 11, 1942, May 14, 1947, and Sept. 28, 1948). He was in the Army for two years shortly before the re-examination in 1947, which was made at the request of the laboratory; the only exposure to loud noise during this time that he could recall was a session of rifle practice, following which his ears "rang for a week." Whether or not this acoustic trauma was responsible for part or all of the increase in impairment that occurred between 1942 and 1947 is unknown, but it is known that further impairment for both of the "border" tones (2,896 and 13,004 cycles) occurred during the next year after the test made in 1947, during which period he worked in an office and was not exposed to acoustic trauma. The last audiogram, for the right ear, is a typical example of "abrupt" high-tone loss, of the sub-category that has 2,048 cycles as the highest frequency well heard.[7]

The left ear of this boy had, at the first examination, a slightly greater average impairment of thresholds for high tones than did the right ear (audiograms of Nov. 9, 1939, in Fig. 3). This situation was reversed by the time the second tests were made, on Jan. 4, 1940 (audiograms not reproduced), and from that time on the impairment of the right ear for high tones was the greater. The progression of the impairment of the left ear occurred somewhat irregularly from the high-frequency end of the scale towards tones of lower frequencies, not by a deepening and widening of a clearly recognized "dip," as in the case of the right ear. At the last examination (see audiogram of Sept. 28, 1948) the impairment for high tones in the left ear was clearly of the "abrupt" type, but the highest frequency well heard was a half-octave higher in pitch than in the right ear.

Two brothers of N. B. also participated in the study of the hearing of Baltimore school children.[10] They were aged 10 and 8 years, respectively, when first examined in the Autumn of 1939, and the younger one, like N. B., had slight impairments of threshold for some of the very high-pitched tones. Like the older brother, the histories of these boys were entirely negative with respect to ear, nose or throat troubles or treatments. None of the three had had tonsils or adenoids surgically removed. Both of the younger brothers had more lymphoid overgrowth of the nasopharyngeal orifices of the Eustachian tubes than did the older brother who was treated with radon, but irradiation was not advised for either of them because their tympanic membranes were rated as only slightly retracted. Because of the large adenoids, however, both brothers were re-examined twice yearly until the study had to be discontinued on account of a shortage of staff after the United States entered World War II. During the period of re-examination (until October, 1941, for one boy and January, 1942, for the other) hearing acuity and conditions of tympanic membranes and of nasopharyngeal lymphoid tissue remained essentially unchanged. Both of these untreated brothers were re-examined in May, 1946, at which time the hearing thresholds of both were essentially the same as they had been in 1939. The tubal orifices of one of them, the younger, were still partially overgrown by lymphoid tissue; in the other boy the nasopharyngeal lymphoid tissue had spontaneously regressed until only a small amount remained and the tubal orifices were both normal in appearance.

The above observations should not be interpreted to mean that the irradiation received by the older brother (see Case 3) caused the hearing impairment for high tones that he developed; *i.e.*, that if he had been handled as were the younger brothers, his marked degree of impaired hearing for high tones would not have occurred. As has been stated in the comments on Cases 1 and 2, I do not believe the real cause of such impairments is in any way affected by either surgical or irradiative therapy. In my opinion, it was in each case merely a coincidence that the treatments were given during the period when the causative lesions, whatever they are, were in the progressive stage. The fact that the hearing impairments of the untreated brothers did not progress, while those of the treated brother did increase markedly, illustrates well, however, the difficulties of prognosis in individual cases.[9]

Case 4: B. M., a white boy, was 13 years old when first examined as a participant in the study of the hearing of Baltimore school children.[10] At the age of 9 years he had had a tonsillectomy and adenoidectomy; otherwise his ear, nose and throat history was negative except for about 2 mild colds per year. Both tympanic membranes were normal in appearance in spite of the fact that the adenoids were large and partially covered the nasopharyngeal orifices of both Eustachian tubes. He heard correctly all the numbers in the phonographic audiometer test, and the tests with a 512 d.v. steel tuning fork also yielded normal data for both ears. With the pure-tone audiometer the thresholds of the right ear for high tones were near the lower limit of the "normal" range. The tests of the left ear revealed a "dip" to the 50 db. level for 5,793 cycles, and some impairment for 8,192, 10,321 and 13,004 cycles; the threshold for the highest tone, 16,384 cycles, was in the normal range for this frequency (see audiograms of Dec. 7, 1939, in Fig. 4).

A nasopharyngeal irradiation treatment, with radon in a brass applicator with a wall thickness of 1 mm., was given by Dr. Crowe on Dec. 18, 1939. When the boy returned five weeks later the adenoids were not so large as previously and both tubal orifices were normal in appearance, but the hearing thresholds for high tones had become worse in both ears (audiograms of Jan. 23, 1940, Fig. 4). His temperature was slightly elevated, but he had no other signs of a "cold." He was not treated this day and was not re-examined again until June, 1940, at which time the hearing acuity of both ears had improved slightly (audiograms not reproduced) and the tubal orifices were still normal in appearance. He was next seen on Dec. 17, 1940, at which time the thresholds for the right ear were as good as at the first test, but the impairment of the left ear had progressed. The "dip" at the frequency of 5,793 cycles, present at the first examination, had become wider in both directions; the threshold for 4,096 cycles had dropped from the 15 db. to the 45 db. level, and the impairments of the thresholds for all the higher frequencies, except 13,004 cycles, had also increased (see audiograms of Dec. 17, 1940, Fig. 4). The tubal orifices were normal in appearance, as they had been since the examination in January, 1940, but the tympanic membranes were now

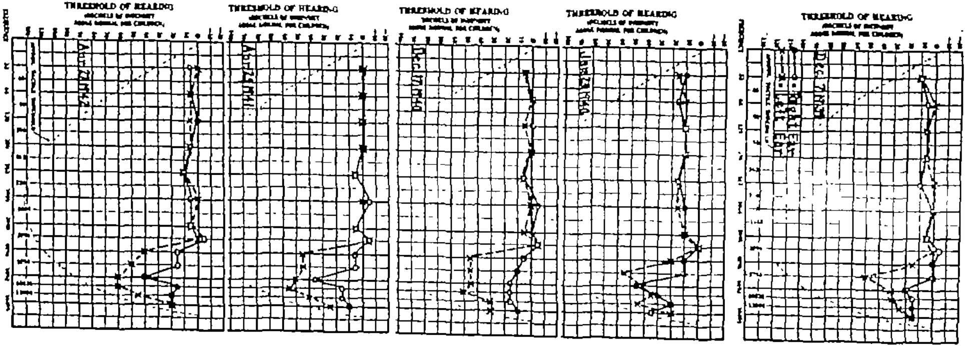

Fig. 4. The changes in hearing acuity for high tones that occurred during 2 years and 4 months in a boy 12 years of age when first examined are shown by the 5 pairs of audiograms, of the 9 made, that are reproduced. Note the good hearing, in this case, for the highest frequency tested, 16,384 cycles per second. See text, Case 4.

rated as moderately retracted instead of as normal. The adenoids were rated as moderate in size and as not having regressed beyond the stage observed in June, so a second radon treatment of the nasopharynx was given Dec. 17, 1940.

When examined 2 months later, Feb. 25, 1941, the adenoids had decreased in size to a rating of small, but the thresholds of the right ear for the four highest tones (8,192 cycles upwards) had dropped to the 30 db. level; the thresholds of the left ear were essentially the same as in December (audiograms not reproduced). At the next examination, made 2 months later, the thresholds of the left ear had changed only slightly, but the thresholds of the right ear for the three highest tones had improved and its audiogram showed a "dip" to the 35 db. level for the frequency of 8,192 cycles (audiograms of April 25, 1941, in Fig. 4). The physical examination on this date was made by Dr. Crowe, who rated the tympanic membranes, the nasopharynx, including the fossae of Rosenmueller, and the tubal orifices as now normal in appearance.

During the next year this boy was re-examined three times (November, 1941; January, 1942; April, 1942). The hearing impairments for the tones from 4,096 cycles upwards persisted in the left ear, as did the "dip" at 8,192 cycles in the right ear. The audiograms of the last test, made on April 27, 1942, are reproduced in Figure 4.

Comments: The period of observation of this boy, B. M., was only a little more than 2 years, a much shorter time than for the other cases included in the present report. It is unusual to have the hearing for the frequency of 16,384 cycles remain so nearly normal when the thresholds for all other frequencies above 2,896 cycles are impaired, and it would have been of much interest to have had further tests of this boy's hearing, to learn not only whether the "dip" nature of the impairment for the left ear changed to a typical "abrupt" type of high-tone loss but also whether the impairment of the right ear progressed by widening of the "dip" at 8,192 cycles, or otherwise. He was in the Navy, however, when the laboratory tried to get him to return for re-examination in April, 1947, and could not come. If his hearing is carefully tested, at some place other than Hopkins, while he is in the Navy or after discharge from it, all of the high-tone loss found will probably be blamed on acoustic trauma, a conclusion that would not be supported by the changes known to have occurred in his hearing acuity between 1939 and 1942. The case illustrates nicely one of the difficulties encountered in the interpretation of hearing tests.[8]

The following case illustrates the usual experience with respect to progression of a slight or a moderate degree of impaired hearing for high tones in childhood, namely, that the thresholds fluctuate a bit but do not change greatly over a period of years. When the first hearing tests of this boy are compared with those of Cases 1 to 4, the case also illustrates the difficulty of predicting, on the basis of the early examinations, whether or not a patient's hearing impairment for high tones is going to be progressive in spite of treatment.

Case 5: This boy, J. M., a private patient of Dr. Crowe, was 9 years old when first examined, Oct. 16, 1939. His audiograms for that date are reproduced at the top of Figure 5. With the phonographic audiometer test he heard the two-digit numbers normally with the right ear but had a 6% loss with the left ear. The sound of the 512 d.v. steel tuning fork was not lateralized from midline point of the head, and the fork was heard for normal times by air conduction and by bone conduction with both ears.

The boy had had several attacks of otitis media between the ages of 2 and 7 years, but none since his adenoids were removed for the second time, in May, 1937. He had had a tonsillectomy and adenoidectomy a few months previously. Nasopharyngoscopic examination in October, 1939, revealed that he again had a marked degree of overgrowth of both tubal orifices by hyperplastic lymphoid tissue. Both tympanic membranes were markedly retracted in all parts and the left one was atrophic and had a healed perforation posteriorly.

A week later, on Oct. 23, 1939, his nasopharynx was irradiated with radon in a brass applicator with a wall thickness of 1 mm.; the dosage given to each side was 1.9 gram minutes equivalent. The treatment was repeated on Nov. 29, with a dosage of 2.29 gram-minutes equivalent to each side. The audiograms made a month later (Dec. 29, 1939) are reproduced in Figure 5. Comparison of the thresholds of the right ear on Oct. 16 and Dec. 29 shows that a considerable drop had occurred in the hearing acuity for high tones, and gives the impression, not borne out by the later tests, that one is dealing here with a condition like that found in the previous cases (Nos. 1 to 4). The nasopharyngeal lymphoid tissue had decreased markedly in amount but the tubal orifices were not quite normal in appearance. The appearance of the tympanic membranes had not changed. He was not treated on this occasion but was given another irradiation treatment, his third, when he returned on March 1, 1940. The audiograms of that date (not reproduced) showed less hearing loss, for the right ear, than in December, but more than at the time of the first test. The left ear had slightly more impairment for high tones than it had in December, but its hearing acuity for low and for middle range tones had improved.

The boy was examined again in June, 1940, at which time, in spite of having but recently had whooping cough, the nasopharyngeal lymphoid tissue had entirely disappeared and the tubal orifices, for the first time, were normal in appearance. The tympanic membranes were as at previous examinations. The hearing (audiograms not reproduced) of the left ear had improved slightly for high tones. When next seen, five months later, the thresholds of the left ear for high tones had again improved slightly and the thresholds of the right ear were in the normal range (see audiograms of Nov. 6, 1940, Fig. 5), but there was some lymphoid tissue present in both fossae of Rosenmueller and the posterior lip of

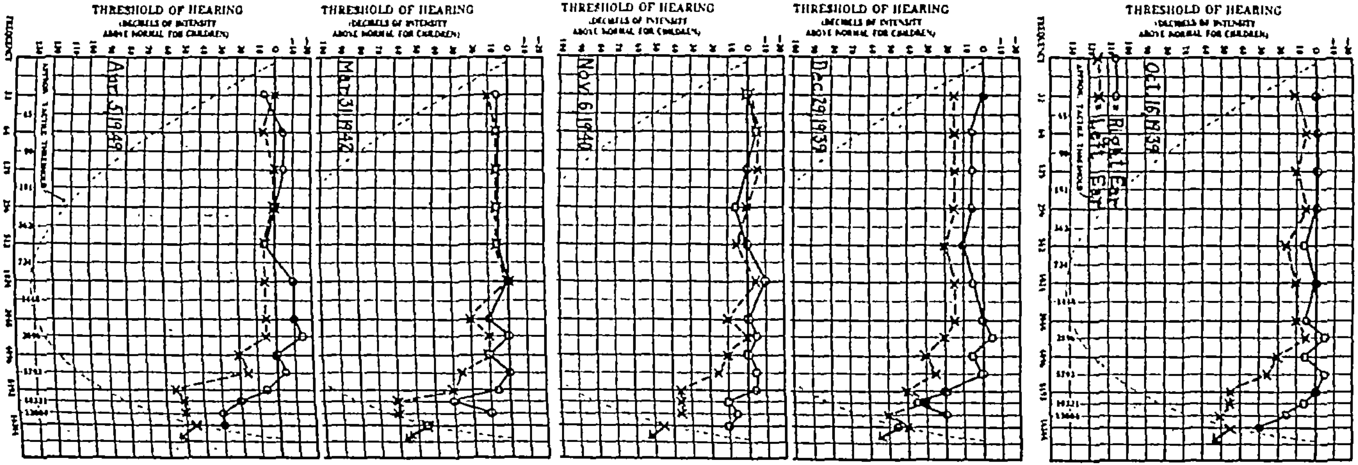

Fig. 5. This series of 5 pairs of audiograms, of the 13 made during a nearly 10-year period of observation of a boy who was 9 years old when first examined, illustrate the usual experience of children who have slight to moderate impairments of thresholds of hearing for high tones, namely, fluctuations of the thresholds but only a very little permanent progression of the impairment See text, Case 5.

the right Eustachian tube orifice was red and granular in appearance. He was given his fourth irradiation treatment this day, and a fifth treatment was given on Feb. 17, 1941, at which time the orifice of the right tube was still red and granular and the hearing acuity for this ear for very high tones had dropped slightly (audiograms of this date not reproduced). The lymphoid tissue on the lateral walls of the pharynx, particularly on the right side, which had been noted at the earlier examinations, had increased in amount, and it also was given an irradiation treatment on Feb. 17, 1941.

When the boy returned nearly a year later (Jan. 28, 1942), the hearing acuity of the left ear for high tones was worse than in any of the audiograms reproduced in Figure 5, the orifice of the right Eustachian tube was still not normal in appearance and there was much hypertrophied lymphoid tissue on the lateral walls of the pharynx, particularly on the right side. Thresholds for the right ear had dropped only slightly; the readings for the three highest frequencies were 25, 20 and 35 decibels, respectively. An irradiation treatment was given to the right side of the nasopharynx and to the right side of the pharynx.

Two months later, when next seen, the pharynx and the nasopharyngeal orifice of the right tube were so normal in appearance that he was not given another treatment. The audiograms made at this time are reproduced in Figure 5 (audiograms of March 31, 1942). He was not seen again until Feb. 6, 1943, at which time the nasopharynx and the pharynx were still normal in appearance, but he complained of a cough which he said had bothered him since he had whooping cough in the Spring of 1940. No cause for the cough was found. Special hearing tests were not made on this occasion, but when he was seen in October, 1943, the thresholds for high tones were found to be better, for both ears, than they had been in March, 1942 (see Fig. 5). There was, however, a small amount of lymphoid tissue present in both fossae of Rosenmueller and he was given another irradiation treatment, his last (as of the date of writing), to both sides of the nasopharynx and to both lateral walls of the pharynx. The dosages were: 2.3 gram-minutes equivalent, with radon in the 1 mm. thick brass-walled applicator, to each side of the nasopharynx, and 1.9 gram-minutes equivelent to each lateral wall of the pharynx. At this time he had had no ear infection since more than two years before his first irradiation treatment, and for the past three years he had had only about one cold per year, each of which was of brief duration.

When he was next seen, in June, 1945, the boy reported having had no colds the previous winter; his nasopharynx and pharynx were in good condition and his hearing was better than it had been at any previous examination. Even for the left ear the only impairments of thresholds were for the three frequencies above 8,192 cycles. In September, 1947, the physical conditions and the hearing thresholds were about the same as in 1945. By April 5, 1949 (see audiograms of that date, Fig. 5) the hearing thresholds for high tones had again dropped to levels that average slightly worse than he had at the beginning of the period of observation, nearly ten years previously. He complained now of a chronic post-nasal discharge, which he said was of several years' duration. He was found to be sensitive to dust, cats, and feathers. No evidence of sinus infection was found and the nasopharynx and the pharynx were both entirely free of lymphoid tissue. The tympanic membranes had not changed materially in appearance since the first examination of them was made.

DISCUSSION AND CONCLUSIONS.

The facts presented above show that the "abrupt" type of high-tone loss can, and sometimes does, develop or progress markedly during childhood, and that the development or the progression of this type of impaired hearing may occur gradually, over a period of years.

The evidence further shows that for the cases reported neither of the usual diagnoses with respect to the remote or true cause of this type of hearing impairment would be correct. A history of exposure to acoustic trauma of the blast type could be elicited in only one of the four cases, and in this instance the exposure occurred after a major portion of the boy's hearing impairment had developed. The other of the two usual diagnoses, namely, congenital cochlear lesion, malformation or failure to develop, obviously would be incorrect for any of the cases cited, except in the sense that the diagnosis might imply inheritance of a tendency to develop a cochlear lesion of the basal turn long after birth. This interpretation of the term congenital naturally cannot, on the basis of the available evidence, be ruled out of consideration as the possible remote cause of the impaired hearing that occurred in these children while they were under observation.

Either to prove or to disprove that the tendency to develop this type of hearing impairment during childhood is inherited, it would be necessary to have available, for the corresponding years of their lives, similarly detailed data with respect to the hearing acuity of the relatives of the children who have participated in the follow-up studies reported. Observations with respect to the present status of the hearing of the parents and of the other adult relatives of the children would not suffice to decide this question. In this connection, it should be recalled that Ciocco's data,[1] cited in the opening paragraph of this paper, indicate that about 60 per cent of adults have some form or other of this type of hearing impairment.

The high incidence of "abrupt" high-tone losses in adults, and especially the great difference in the incidence of this type of hearing impairment in children and in adults (see above), makes it seem probable that a follow-up study of the hearing acuity of a large number of persons, covering the age period from childhood to middle age, would reveal numerous examples like those reported in this paper. It seems improbable, in view of the evidence now available, that exposure to acoustic trauma of the blast type accounts, together with possible congenital lesions, for the high incidence of "abrupt" type high-tone losses in adults. A follow-up study such as suggested would of necessity take many years to complete, and would require the long-time cooperation of many persons. Such a follow-up study would, however, doubtless yield basic information about the causation of the most common form of impaired hearing that occurs in man. The study would also afford a better basis than now exists for clinical prognosis and for advice to patients who have this type of hearing impairment. To make such a study of a significantly large sample of the population, making allowances for the inevitable disappearance of many of the cooperating subjects, would require financial support of considerable magnitude.

As was stated in the comments on Case 1, the evidence available with respect to the examples reported does not support the idea that infection, either bacterial or viral, is commonly the remote or true cause of the "abrupt" type of high-tone loss. Such a hypothetical cause is not completely ruled out of consideration by the available evidence, however, primarily because the number of cases that have been observed during progression of the impairment is small. The crucial evidence needed to determine this point, as well as others, could be obtained by analysis of the observations that can be made in a lengthy follow-up study such as indicated in the preceding paragraph.

The development or the progression of "abrupt" type high-tone losses is not causally related to nasopharyngeal lymphoid tissue near the tubal orifice, or elsewhere, so far as the observations of the present paper indicate. In each of the cases reported shrinkage of the hyperplastic lymphoid tissue present at the beginning of the period of observation was successfully and promptly accomplished by means of irradiation with a radon applicator. Progression of the impairment of hearing for high tones continued, however.

That the treatment itself caused the development or the progression of the impairment seems extremely improbable, in view of the known high incidence of this type of impaired hearing in adults before the days of nasopharyngeal irradiation, and in view of the additional fact that most of the children who have had the irradiation treatment have not, during a period of time similar to that of the present follow-up study, developed or had a marked progression of an "abrupt" type of high-tone loss.[9] Any causal relationship that may appear, from perusal of the case reports of the present paper alone, to exist between nasopharyngeal irradiation and "abrupt" type high-tone loss should be interpreted in the light of the circumstance that the follow-up studies of children have been associated with this method of treatment. In other words, the superficially apparent relationship is most probably nothing more than a temporal coincidence; treatment was given during the age period when the hearing impairment was developing, or progressing, for some other reason, which is as yet unknown, or unrecognized.

SUMMARY.

The "abrupt" type of high-tone loss is discussed from the standpoint, primarily, of remote or true causes of the lesion of the basal turn of the cochlea usually regarded as the immediate cause of the functional impairment.

Four cases are reported, in considerable detail, of children who while under observation either developed an "abrupt" type of high-tone loss or had a marked degree of progression of an already existing impairment of this type. In all four instances, the impairments progressed gradually.

The cause of the impairments, had the last examinations of these children been their first, would have been regarded by most otologists as either an exposure to acoustic trauma of the blast type or a congenital cochlear lesion. The reported observations show that neither diagnosis would be correct for any of these four cases.

Other hypothetical, or even plausible, causes are discussed in the light of the evidence from the reported cases. All are ruled out of consideration, more or less completely, and the conclusion is reached that the remote or true cause of many instances of "abrupt" type high-tone loss is as yet unknown, or unrecognized.

The suggestion is made that a follow-up study of a large number of persons from childhood until they reach mid-adult age would provide basic information about the most common type of impaired hearing that occurs in man, and that such a study would also furnish a better basis than now exists for clinical prognosis and advice.

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TABLE I.

ULTRAVIOLET ABSORPTION OF PERILYMPH FROM DAY OPERATION.

Specimen	Conc. %	D (275)	D (410)	Blood $\frac{D275}{D410}$	Blood Correction (Optical Density)	Corrected E (1% 1 Cm.)
1	2.07	.394	.810	.332	.269	0.060
2	2.68	.632	.665	.513	.340	0.109
3	25.5	.742	.336	.398	.118	0.024
4	15.2	.800	.880	.542	.477	0.021
5	13.8	.393	.298	.410	.122	0.020
6*	1.35	.128	.175	.481	.084	0.052
	2.46	.250	.353	.481	.170	0.054

*Endolymph.

hemoglobin absorption of the corresponding blood sample and comparing it with the corresponding absorption of the ear fluid, the quantitative measure of the amount of contamination was obtained. The peak at 275 was probably due to the tryptophanetyrosine content of the protein present, although a number of other substances such as purines and ascorbic acid absorb appreciably at this wave length and were probably present in the fluid.

The spectrum of spinal fluid showed none of the features of ear fluid. The 410 absorption was very low or absent because this fluid was obtained blood-free. The absorption at 275 did not show a peak, but only a slight shoulder in the rise of the curve to the end absorption at 265. It is evident from the analysis of the two sets of spectra that there are marked quantitative and qualitative differences in the absorbing substances present in the two fluids.

TABLE II.

ULTRAVIOLET ABSORPTION OF SPINAL FLUID.

Specimen	Conc. %	D (275)	D (410)	Blood $\frac{D275}{D410}$	Blood Correction (Optical Density)	Corrected E (1% 1 Cm.)
1	8.40	.092	.005	.332	.001	0.008
2	42.4	.605	.030	.513	.015	0.014
3	100	1.010	.026	.398	.010	0.010
4	100	.990	.277	.542	.150	0.008
5	100	.930	.025	.410	.010	0.009
6	15.7	.177	.077	.481	.037	0.009

The ultraviolet absorption of the fluids is given in Table I and II, together with the extinction coefficients E (1 per cent 1 cm.) calculated from them. Correction was made for the blood contamination.

After ultraviolet measurements were made the total sample of ear and spinal fluids was quantitatively transferred to digestion flasks and digested with Kjeldahl mixture. Because of the large blood dilution (1:1000 or 1:2000), 1 ml. of the blood dilution was digested. The ammonia in the digest was determined by the microdiffusion method of Conway. Correction was made for blood N in the fluids (see Table III).

TABLE III.

TOTAL N.

Perilymph.

Specimen	Weight (Mg.)	Total N Microgram	Blood N N/D 410	Corrected N Mg. %	Blood Correction Microgram
3	44	13	15	28	0.9
4	20	8.1	22	27	2.6
5	18	5.1	17	25	0.6

Spinal Fluid.

Specimen	Weight (Mg.)	Total N Microgram	Blood N N/D 410	Corrected N Mg. %	Blood Correction Microgram
3	133	17	15	13	0.0
4	157	22	22	13	0.9
5	110	16	17	14	0.2

DISCUSSION.

The small number of cases, the minute amounts of inner ear fluids, and the unavoidable contamination of the fluids with microscopic amounts of blood make it necessary to consider the results as not final.

The result of these measurements, however, allow us to draw a number of conclusions. The protein content of perilymph and endolymph as measured by D-275 was more than double that of the spinal fluid in each specimen. Measurements of total N confirmed results obtained with the ultraviolet absorption, thus giving a double check on the results.

The huge difference between the protein contents of the two fluid systems disproves our previous concepts of identity of perilymph and cerebrospinal fluid. In addition, a free flow of spinal fluid into the cochlea does not appear possible in view of the definite difference in chemical structure of the two fluid systems. Measurements of Na content were obtained in one of these cases and showed it is present in almost identical concentration in both spinal fluid and perilymph.

The results give additional support to the existence of a barrier membrane[1] at the cochlear opening of the aqueduct. This membrane apparently does not permit passage of large molecules of colloids like proteins. Equal concentration of Na on the two sides of this membrane, on the other hand, is strongly suggestive of a semipermeable nature of the barrier membrane.

Experiments performed by Gisselsson[3] and Altmann and Waltner[2] showed that crystaloids diffuse through this membrane in monkeys, cats and rabbits.

Perilymph probably is derived from at least two sources. One of these is the cerebrospinal fluid which supplies some of the crystalloids like Na, etc., which are able to diffuse through the membranous barrier. A similar diffusion of crystalloids equally may take place through Reissner's membrane from the endolymph into the perilymph. Experimental evidence exists in cats and rabbits showing an easy passage of iron salts through intact Reissner's membrane.[2] Identification of the source of proteins and other large molecular components of the perilymph still awaits additional evidence.

Protein content of the endolymph studied in one single case of Ménière's disease revealed that it is in the same range as the perilymph. This single specimen, although two measurements gave identical results, would have little significance alone except that it fits very well into the picture pieced together with the help of animal experiments (cats) by Aldred, Hallpike and Ledoux[4,5] which demonstrated the almost identical osmotic pressure and index of refraction of the endo- and perilymph.

A large series of experiments on cats were performed by us, using the same method of measurement as on human material. They allow us to make the same conclusions on the protein content of the perilymph. These studies will be published in a separate paper.

It still has to be seen whether these conclusions can be applied without change to the normal perilymph, or are valid only for perilymph and endolymph in Ménière's disease.

With perfection of the method of collecting samples of perilymph it may become possible to obtain perilymph from a larger number of suitable cases of otosclerosis during fenestration operation without impairing the function of the ear.

SUMMARY.

1. Five specimens of perilymph were obtained from the lateral semicircular canal of humans while performing Day-operations for Ménière's disease.

2. One specimen of endolymph was collected from a patient with Ménière's disease during a Portmann operation.

3. Measurements of the ultraviolet absorption and total nitrogen were made on these fluids.

4. The protein content of the inner ear fluids as indicated by the above measurements was more than twice that of the cerebrospinal fluid of the same patients.

5. These figures corroborate the existence of a barrier between perilymph and spinal fluid, probably of semipermeable nature.

Thanks are expressed to Mr. Herbert Wohl and Mr. Robert Franzl for technical assistance.

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180 Ft. Washington Avenue.
630 W. 168th Street.

AN EXPLANATION OF CERTAIN TYPES OF TINNITUS AND DEAFNESS.*

(Lantern Demonstration.)

EDMUND PRINCE FOWLER, M.D., and
EDMUND PRINCE FOWLER, JR., M.D.,
New York, N. Y.

During the last one hundred years the advances in medicine have brought us to a stage where we now can examine properly prepared tissue under high magnification, and see microscopic morphological changes resulting from disease. We also have learned much about the infectious diseases and something about why they cause disturbances of the tissues and many ways in which they may be combated and cured. We know that mechanical and acoustic traumas and poisons as well as certain diseases may and frequently do cause tinnitus and deafness. We know something about the inheritance of deafness. We have studied the incidence of tinnitus in the different forms of ear disease causing deafness. We know how to estimate the timbre of the tinnitus, and measure its loudness. We know how to measure the hearing capacity and have discovered several useful tests for aiding differential diagnosis in deafness and in tinnitus; but there are many instances in which deafness or tinnitus or both occur without any history of a prior inflammatory episode, without a family history, without any acoustic trauma, any fatigue or poisoning, or any abuse of drugs; and at autopsy, if we are fortunate enough to obtain one, we view only the end-results, we learn nothing as to causation.

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*Read at the joint meeting of the Section on Otology, New York Academy of Medicine, and the New York League for the Hard of Hearing at the latter's Fortieth Anniversary celebration, Roosevelt Hotel, New York, N. Y., April 19, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 17, 1950.

Most people with deafness and tinnitus appear otherwise perfectly healthful. They seldom reveal vitamin deficiencies, or obvious disturbances in their mucous membranes, eyes, locomotor or cardiovascular systems. They often suffer from no apparent disease whatsoever. Maybe the only detectable abnormality is that one ear is deafened, or entirely deaf, or that there are head noises in one or in both ears. In some instances both ears are severely deafened (even totally). People who suffer from deafness associated with severe tinnitus are usually very sensitive, tense or "nervous." They are often called "the ulcer type." It is this mysterious tinnitus and deafness often seen, the origin of which has up to now eluded medical science, which we will discuss in this paper, and for which we believe we have discovered a cause, a cause which should be preventable, and even controllable. Please note that we say "a cause," not "the only cause."

If deafness is sudden and total it is customary to "guess" that it is due to a vascular accident. By vascular accident is meant a hemorrhage or bleeding into the tissues from a ruptured blood vessel. Without autopsy vascular accidents are difficult to prove; in fact, rarely have been proven in these cases. Moreover, in the types of deafness we are here discussing the deafness may not be sudden, and generally at least for a long time not total. Such patients as a rule show no sign or symptoms of disease or loss of function of any kind in any of their sense organs except the ear, in spite of the fact that many of them have been examined at the time or immediately after the tinnitus or deafness was first noticed. At one time or another they may have had vertigo, nausea and vomiting, but this may always be traced to the vestibular end-organ. The majority of these patients are not old persons, although these symptoms are not uncommon in old people. Arteriosclerosis, though it is sometimes present, is probably not prominent in the etiology. Arteriosclerosis frequently occurs without any tinnitus and deafness. These types of tinnitus and deafness also occur in people with various other bodily disorders and diseases. The underlying etiology for the disorders in other organs is often the same as that in the ear.

For instance, in our experience sudden deafness and tinnitus occur quite frequently with severe liver disturbances, and with duodenal ulcers and ulcerative colitis.

The number of discernible factors underlying and conceivably common to many diseases as well as to various disturbed psychosomatic states in otherwise healthful people is limited. What are some of the most commonly present factors which deserve consideration? Eliminating undiagnosed inflammatory destructions, poisons, blood dyscrasias, and trauma, there remain outstanding only the neurovascular, psychic, and nutritional disorders. The last may be disregarded as a common factor here because they are rarely encountered in these people, at least to no greater extent than in those with no tinnitus and no deafness. It is impossible to separate the psychic and neurovascular factors because they are often closely integrated, and, therefore, they should be, and herein will be, considered together. The question then is, how can psychoneurovascular factors cause the type of tinnitus and deafness we are here considering?

The general conception of the effects of emotional disturbances is that they may complicate the symptomatology of any disorder by their effect upon the psychological behavior of the patient, but that any effects that they may have upon the tissues of the body are transient and of no importance from the standpoint of a primary cause of disease, *i.e.*, that psychical disturbances in the average patient are "overlays" rather than the cause of disease or symptom. We believe this concept is not in accordance with the facts.

What do we mean by "emotion"? The dictionary definition is misleading. It takes no cognizance of unrecognized (subconscious) emotional states. Strictly speaking, emotion is a psychological and bodily state that in greater or less degree accompanies all mental and physical activity. Even slight emotion is accompanied by a physiological change. When it is extreme we can recognize it by flushing or blanching of the face, dilatation of the nares, trembling, weeping, a display of temper, hysterics, etc. We believe with Wolf, Cannon, Lewis and others that there are often present less obvious changes in the circulatory, respiratory or muscular systems.

Psychologically speaking, there is an "effect" or "feeling tone" that accompanies all sensation. When the "feeling tone" is exaggerated by ideation (thoughts or ideas, conscious or unrecognized) it is called "emotion."*

Failure to act or overacting emotionally to the environment are factors which have been shown to lie at the root of many so-called "functional disorders." Also they may be largely responsible for the time of onset, severity, duration, recurrance, variation, and ultimate outcome of various diseases, and even for the instigation of many pathological states.

Profound changes occur in both man and beast from fear, rage, excessive fatigue, insecurity, longings, unhappiness, bereavement, despair or frustration through activity of the autonomic (involuntary) nervous system; both the parasympathetic and the sympathetic divisions are involved in these changes.

Under emotional stress blood pressure variations, flushing of various areas of the skin, blanching, fainting, nausea and vomiting and involuntary dilatation of the sphincters are observed, and are largely parasympathetic reactions. The involuntary or autonomic nervous system, whether activated directly or through the mediation of the blood stream, can affect the functions of almost every tissue or organ in the body whether it be in health or in disease, and whether or not the blood pressure is high or low, or average normal.

Both efferently and afferently there are intimate connections between the autonomic and central nervous systems. The hypothalamus is the chief integrating center for the autonomic impulses, and it has connections with the thalamus and the cerebral cortex. The cerebral cortex influences the autonomic nervous system in both of its divisions, and both divisions are widely distributed in the body; to the skin, blood vessels, viscera, reproductive mechanisms, secretory and excretory organs, endocrine glands, and the sense organs.

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*This definition of emotion was evolved after consultation with Dr. L. Vosburg Lyons, of New York, N. Y.

The ultimate reaction of so-called emotional episodes upon the tissue cells is caused by the chemical mediators adrenalin and sympathin called forth by sympathetic stimulation, and by acetylcholine released by parasympathetic action, and also by the hormones of various glands of internal secretion either directly or indirectly.

Whether the resulting clinical symptoms or lesions are more sympathetic or more parasympathetic depends upon the relative stimulation of these two divisions of the autonomic mechanisms, and upon the hyper- or hypoactivity of the tissue cells, or the nerve supply to the organ affected.

The psychosomatic effects of emotions upon many disorders and diseases are legion, but when an incidental illness coincides in time with severe emotional shock the illness is made worse and a relapse occurs, and the two together may be more than the patient can stand.

Frequently the emotional factors are overlooked because the physician assumes that the patient will bring them to his attention if they are important. As a matter of fact we all of us often have more powerful "forgetories" than "memories," and so we do not bring them to the attention of our physician.

Old memories aroused by some appropriate stimulus can bring back the intense emotions originally associated with them. Such memories may be subconscious and still bring back the emotional reactions and their concomitant repercussions upon the autonomic neural mechanisms. This may occur during sleep. The patient may then attribute his disturbed feelings to some recent happening, and not realize its true source, an episode in the past. When there is an autonomic hypersensitivity, or an already damaged organ, the renewal of emotional tension or imbalance can lead to exacerbations of the lesion and to an increase in symptoms.

How may we decide whether or not emotion is a significant factor in "organic," or in so-called "functional disturbances" of the ear? Of course one preliminary is to exclude prior organic disease of the auditory apparatus. This is often difficult, and moreover both organic and functional disorders may arise from similar emotional states, and both may exist at the same time.

Important criteria for correlating emotional and somatic factors are:

1. Establish a time relationship between the emotional episodes and the onset or exacerbation of symptoms. (We are herein concerned with tinnitus and deafness in otherwise apparently healthy persons.)
2. If no correlation is remembered or recognized, or no memory remains of the emotional upsets, yet the patient is and has been hypersensitive to emotional stimuli, then it is highly probable that emotion was a factor in the etiology. There is *nothing else more probable.*

How do episodes of hypersensitivity, or as they are commonly called, "states of emotional instability," interfere with normal metabolism and bring about irritation, diminished function and even degeneration of the auditory (and vestibular) neural mechanisms? An outline of what often probably happens may be set forth as follows: Whether as a result of a present or prior illness hypersensitiveness of an organ results in an over response to sympathetic stimulation. The blood vessels in the organ over contract, or over relax, during too long periods of time. Over contraction cuts down the calibre of the small arterioles and venules, slows the blood stream and furthermore it has been shown that with sympathetic stimulations blood sludge is created. This further interferes with the passage of blood through the swollen vessels. If severe these reactions may shut it off completely from a given area.

The stasis deprives the tissues of oxygen, sugar and other metabolites, and interferes with the removal of waste and heat from the tissues. Oxygen is one of the most important blood borne materials, and even a slight local oxygen deficiency will upset the healthy functioning of the tissues and eventually cause degeneration in the more susceptible elements therein. Unless collateral circulation promptly comes to the rescue the following events have been observed to take place:

Decreased blood volume alone, even following a loss of 300 to 500 cc. in blood donors, is sufficient to cause tight contractions in first one and then other sets of arteries, arterioles, capillaries, postcapillaries and venules observed in the ocular conjunctiva. Only a few larger vessels may remain open. Similar reactions occur in the blood vessels of other tissues. There may be the initial signs of shock, such as profuse sweating, chilliness and giddiness. If no counter stimuli or inhibitions intervene prolonged contractions have been observed, and the affected tissues are literally asphyxiated for considerable periods of time (see Fig 1).

After traumatic shock (even without loss of blood) and in all severe infections and toxic diseases, thick agglutinated masses have been observed to form by the sticking together of the erythrocytes (red blood cells). The masses are called "sludges." Inserting a fairly large needle into the body is sufficient to produce sludging of the blood, even causing the patient to react to a slight surface irritation (*i.e.*, electric shock). Even a provocative remark may cause a contraction of the smaller vessels often followed by sludging of the blood.

Sludges eventually find their way into the general circulation, and before they are destroyed, as eventually happens in the liver and spleen, they may have caused profound changes in some areas of the body. They are most easily observed in the smaller arteries and venules in the ocular conjunctiva of the unanesthetized patient and here provide us with a fair sample of all of the circulating blood.

Using binocular microscopes with high magnification (50 to 100×) and brilliant illumination obliquely directed, with adequate thermal protection, we can focus our microscope on the vessels in the bulbae conjunctiva to study the normal and pathological blood flows. We have been intensely studying the degrees of sludging, its rigidity and retarding effects upon

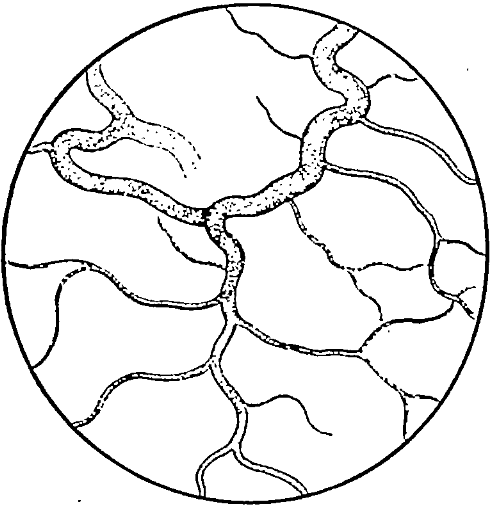

Fig. 1. Sketch of a normal conjunctival field × 100 approx.

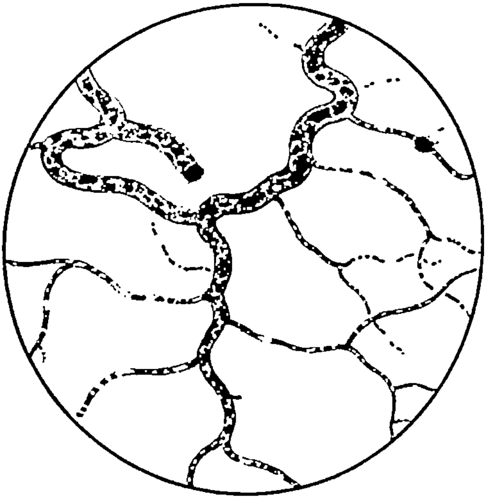

Fig. 2. Sketch of the same conjunctival field after cervical sympathetic trunk stimulation or injection of intravenous epinephrin. The smudge, upper right, is a small hemorrhage.

Reprinted from the Proceedings of the Society of Experimental Biology and Medicine. Blood Sludge from Sympathetic Stimulation, by E. P. Fowler, Jr., 72:592, 1949.

the circulation, the state of the vessel walls, and their pathologic reactions to stimuli.

Knisely* has demonstrated that in perfectly healthy persons (and in the so-called "lower animals") the blood flow is normally laminar and streamlined. No stacking (Rouleaux) or clumping of the blood cells was obeserved, except in abnormal conditions.

In health, and if no local trauma has occurred, the red cells are not coated with a sticky precipitate and show no tendency to adhere to each other. Neither white nor red cells adhere to the smooth and clean inner walls of the small vessels. Around the axial stream of cells is a peripheral concentric layer of blood plasma. Each succeeding layer toward the periphery flows more slowly than the one immediately inside it, and each layer is exactly one red cell thick. The diameters of the narrowest and more powerfully contractile vessels in the tissues (the arterioles, terminal arterioles, contractile sphincters, etc.) may remain constant or change from moment to moment as is necessary for physiological adjustments in response to changes in the equilibrium between "constrictor and dilator" substances reaching the vessel wall from *1*. blood within the vessel, *2*. cells surrounding the vessel, and *3*. nerve endings, in, upon, or near the vessel wall. Temperature changes in the surrounding tissues also affect the contractile elements.

Further, according to Knisely and his co-workers, every homeostatic dilatation of any group of small vessels produces hydrodynamically an increase in the rate of blood flow through these vessels and an increase in the rate of supply of oxygen and metabolites, and in the removal of wastes.

The blood normally flows so rapidly in most arterioles and venules (diameters 60 to 120 μ) that the individual red cells cannot be seen. This invisibility serves as a fairly useful criterion for judging the adequacy of the speed of flow through most of the open-tissue capillaries. If unagglutinated blood cells cannot be seen under high magnification there is no visible hemoconcentration taking place in the capillaries. In other words, the capillary walls are not leaking blood fluid.

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*Knisely, M. H.; Eliot, T. S.; Block, E., and Warner, L.: Science, 106:2755, 1947.

Every blood cell, red and white, passing through open arteriole terminals is forcibly distorted because it cannot be shoved through without being elongated or folded. In the capillaries there is more room, because the diameters vary from a little less than one to as much as at least two and one-half times the diameters of the red cells, and true capillaries can usually dilate without losing their tonus.

Knisely believes that almost every arteriole to capillary or sinusoid to venule pathway can contract tightly shut throughout its length, its lumen then being zero, and that when no blood can enter, in some or all of its parts dilatation may take place to the maximum permitted by the surrounding supporting connective tissues, normally not over two and one-half times the diameter of the red cells. Forcibly overdilated capillaries permit both blood plasma and protein to leak into the sourrounding tissues. *No severely ill patient has been observed who did not have sludging of the blood and visibly damaged vessel walls.* "Under all conditions, in health and in disease, the arterioles and capillaries are a perpetual bottleneck in the vascular system. These bottlenecks are increased by the resistance of sludged blood to its own passage, and the rate of flow is forcily reduced in all the capillary beds of the body."

In some organs there is such a wide margin of safety that moderate contraction of the vessels or moderate sludging causes no detectible symptoms, but in the auditory neural apparatus of the cochlea the vessels are terminal, there is no collateral circulation to come to the rescue when a branch of the cochlear artery is obstructed. It is reasonable, therefore, to suggest that contractions or sludging or both could be, even often are, disastrous to the neural elements involved. It should not be surprising if irritation so caused resulted in tinnitus, and if prolonged eventually resulted in an irreversible degeneration in the neural tissues; the auditory ganglion cells, the nerve fibres, and their insulating sheaths. Such degenerations are regularly found postmortem in patients with these types of tinnitus and deafness. It should not be surprising that emotional episodes, remembered or forgotten, suppressed or evaded, are usually a determining factor in emotional problems, and that they can set up the reactions described. We have frequently been permitted to correlate them with the onset of, and with variations in tinnitus and deafness.

We wish to point out that vessel contraction, sludging, and the accompanying phenomena can affect not only the neural elements in the ear (and elsewhere in the body) but also nonneural tissues, even the bones, and here we have a promising lead for investigations in connection with the etiology of otosclerosis.

Sludge is encountered in many diseases and disorders of the body. How does it happen that it is not always accompanied by tinnitus and deafness? One reason is that there are many kinds of sludge, and sludging phenomena, and varying time factors involved; moreover, the prior presence of sludging, its degree and the prior stability and health of the organ are of importance. In studying the vessels and the sludging phenomena, the size, shape, consistency, compressibility, spacing, and rate, and continuity of flow are important. Heredity and previous trauma from injury or disease will determine whether sludge lingers and produces local disorders, or pours through the vessels freely to organs where microscopic blocks may produce no symptomatology.

Now that we have learned something about the phenomena accompanying sludging we know in which direction our investigations and treatments should be directed. We already possess one means for controlling the phenomena, and more will be forthcoming. They will be discussed in a future report.

It is high time that the otologist should not be satisfied with a diagnosis of the pathology, or of the infecting agent, or of the mechanical cause of an ear disease, or even with a study of dead tissues under the microscope. He should investigate the biological causes which made the patients susceptible to them. He should study pathology in the living patient. The closer he gets to a study of living individual cell physiology and pathology the nearer he will get to the truth.

We feel that the observation of changes in the blood itself in cases of deafness and tinnitus, and the alleviation of tinnitus by intravenous procaine (which had previously been shown to break up blood sludge) are steps in the right direction. We have, of course, not proven incontrovertibly that emotionally induced blood sludge produces tinnitus and deafness, but there certainly seems to be strong evidence that this may be the case in some individuals.

THE CYTOLOGIC DIAGNOSIS OF PRIMARY BRONCHOGENIC CARCINOMA.[*][†]

JOHN J. O'KEEFE, M.D.,
Philadelphia, Pa.

In the universal endeavor to eradicate cancer, the literature[6,7,11] is abundant in its recordings of efforts directed towards the development of better diagnostic methods. The motivating force being that only by the establishment of such procedures will early diagnoses be feasible and, consequently, cures possible.

The summation of facts gathered from the recent literature[1,3,12] indicates that primary bronchogenic carcinoma is predominantly a disease of white men; its greatest incidence is between the ages of 45 and 60; it has a definite predilection for the right lung; the average length of life in cases in which the patient is not amenable to surgical cure is 12 to 18 months. Its cardinal symptoms, in the order of frequency of occurrence, are recorded as cough, pain in the chest, expectoration and dyspnea. The histopathologic picture presented is predominantly that of epidermoid carcinoma, with an occasional report of adenocarcinoma.

Clinical manifestations of this disease usually are not evident until after the primary lesion has undergone moderate growth. The majority, of squamous cell type, manifest symptoms through invasion of regional tissue, producing secondary factors, such as erosion and ulceration of the mucosa, bronchial obstruction with retained secretions, secondary infection, atelectasis and/or compression of one of the larger pulmonary vessels. Tumors originating in the extreme periphery,[4,9] the "alveolar cell tumors," are more apt to produce

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[*] Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

[†] From Department of Bronchoesophagology, Jefferson Hospital, Philadelphia, Pa.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1950.

symptoms referable to the pleura, or to remote organs by metastases. Too often, both types produce metastatic lesions before adequate consideration has been given the symptoms caused by the primary lesion of the lung.

TABLE 1.

SYMPTOMS ON PATIENT'S ADMISSION TO THE HOSPITAL.

	Per Cent
Cough	76.3
Thoracic Pain	62.5
Sputum	56.4
Dyspnea	39.6
Hemoptysis	38.9
Loss of Weight	36.6
Loss of Strength	32.0
"Cold"	26.7
Night Sweats	7.6
Hoarseness	7.6
Wheezing	2.2

The development and presence of detectable physical signs are evidences of advanced growth and almost invariably preclude the possibility of operability.

The literature varies somewhat in its recorded statistics,[2,3,5,9,12] but such variability only lends emphasis to the facts *1*. that the symptoms of early bronchogenic carcinoma follow no stereotyped pattern, *2*. that every symptom referable to the chest reported by the patient should be considered as organic in origin, and *3*. that investigation should be pursued until an accurate diagnosis is attained.

DIAGNOSTIC METHODS.

Roentgenology: The Roentgenogram is probably the most valuable single means of obtaining an early presumptive diagnosis of bronchogenic carcinoma. It is well to bear in mind that early developing densities in the lung fields follow no form or pattern; their outlines are indistinct and their margins irregular; small shadows, situated peripherally, with a tendency to clear, only to recur, are in all probability neoplastic; and that the commonest Roentgenologic manifestation of total bronchial obstruction[4,10] is atelectasis of the segment, lobe or lung tissue distal to the tumor. The adjunctive use of

employed in order to secure an earlier positive histologic diagnosis.

Aspiration Biopsy: Although approved of initially, the associated danger of implant metastasis in the tract of the aspirating needle has fostered much controversy as to the practicability of its continued use.

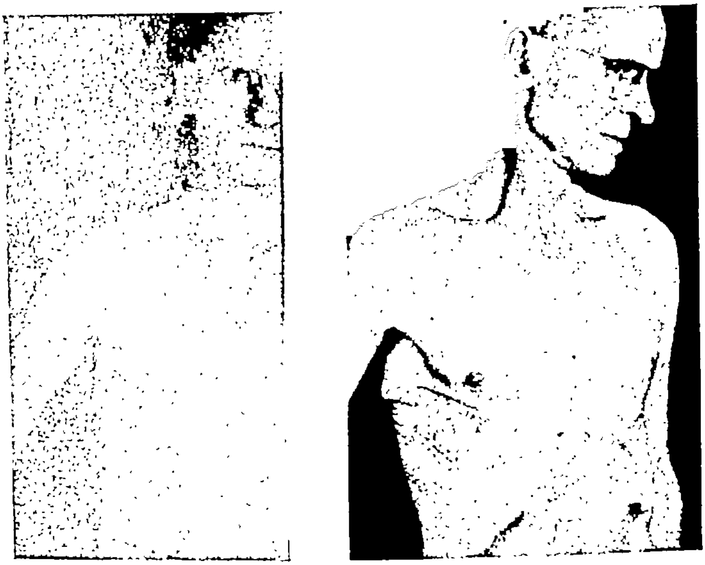

Fig. 2.

A.
Photograph of pneumonectomized patient, showing development of implant metastases in the chest wall at the exact site of aspiration for biopsy.

B.
Photograph of the patient after excision of the lesion.

Exploratory Thoracotomy: To balance the scale, the practice of exploratory thoracotomy has gained universal widespread favor; however, even this practice has its shortcomings, for at the time of thoracotomy the nature of a small, centrally placed lesion is indistinguishable from an area of pneumonitis, an abscess or an anthracotic or silicotic nodule.

Cytologic Method: Most recently developed, and of excellent promise, is the practice of cytologic study of bronchoscopically removed secretions. This procedure has added a

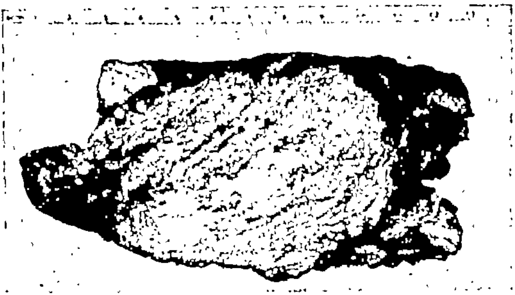

FIg. 2. C.
Photograph of the tumor removed from the chest wall.

high percentage of positive results in the early diagnosis of bronchogenic cancer. It is of particular value in the study of those lesions that are peripherally situated and bronchoscopically inaccessible. Its practice, too, is free of the dangers, real or imaginary, that are integral parts of the technique of aspiration biopsy and thoracotomy.

CLINICAL STUDY.

The stimulating influence in the development of this method of diagnosing carcinoma originates in the staining technique as devised by Papanicolaou.[11] Its application to carcinoma of the lung was initially reported by Herbut and Clerf,[7] in 1946. These observers, reporting on 30 consecutive cases of lung cancer, state that "cancer cells were demonstrated in 22 cases, or 73 per cent. In the same series a positive morphologic diagnosis, from a study of tissue removed endoscopically, was obtained in 11 cases, or 36 per cent. Cancer cells were present in secretions from seven cases in which bronchoscopy was negative."

Consideration and study of this report strongly suggests that in the endeavor to diagnose cancer early, a method of singular merit has been devised. Further, it leads one to query, "Will the diagnosis so obtained be early enough to

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THE
LARYNGOSCOPE.

VOL. LX | OCTOBER, 1950. | No. 10

THE MANAGEMENT OF DEAFNESS.*

KENNETH M. DAY, M.D.,
Pittsburgh, Pa.

Deafness, in greater or lesser degree, is one of the most common afflictions of the human body at some time during life. Until the past generation it was one of the most neglected of human ailments. We can be justly proud of the progress made in this field of medicine in recent years though there is still a long way to go.

Congenital hearing defects should occur more rarely with correction of Rh incompatibility and with better control and prevention of such diseases as syphilis and German measles in pregnant women.

Toxic damage of the cochlea or acoustic nerve as a sequela of the acute exanthemata is also on the decrease due to effective prevention and efficient management of the primary disease. We no longer see the ravaging effects of diphtheria and typhoid fever because the primary diseases have been nearly eradicated. Toxic nerve deafness still occurs in some cases following severe attacks of measles, scarlet fever, mumps and whooping cough though these also are on the decline. The acoustic nerve is sensitive to certain drugs such as quinine,

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*Wherry Memorial Lecture presented at the Fifty-fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 11, 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 29, 1950.

streptomycin and occasionally the salicylates. With more careful and judicious usage of these drugs toxic nerve damage has been greatly reduced.

Deafness due to destructive local lesions from middle ear and mastoid infections has decreased tremendously in the past decade with the advent of biochemicals; in fact, acute mastoid infections now have become almost a rarity.

The prevalence of chronic suppurative otitis media with cholesteatoma has not been noticeably affected by use of the biochemicals which have little or no effect on this condition. Resort to radical surgery is needed just about as frequently now as it was a generation ago; however, there has been a definite improvement in surgical judgment and technique. Whereas, formerly, the classical radical mastoidectomy usually was performed for this condition, most otologists now modify the operation to suit the individual case rather than make the case fit the operation. They now preserve the eardrum and ossicles whenever feasible, thus preserving the hearing at a higher postoperative level. The basic cause of attic perforations with subsequent suppuration and the development of cholesteatoma, as yet, has not been satisfactorily explained and continued study of this condition is needed. It is my opinion that perforations of Shrapnell's membrane occur most frequently as a complication or sequela of chronic secretory otitis media. During the past five years I have observed attic perforations occur in four cases of recurrent secretory otitis media in which the eardrums previously had been intact. These four cases developed secondary cholesteatomas, and two of them required surgical intervention. I also have seen perforations of Shrapnell's membrane occur and later close spontaneously if secondary infection did not occur. Open, dry perforations of Shrapnell's membrane with no history or evidence of infection are not rare and some of these cases subsequently develop otitic infections and cholesteatomas, the most common cause being the introduction of water into the meatus from swimming or syringing the ear.

Secretory otitis media or otitis media with effuson is a condition far more prevalent than is generally realized, being especially common in children. I do not agree with some of my confreres that these so-called fluid ears are occurring more frequently in the past few years than formerly. I maintain that they have been present continuously and were not recognized by many otologists. This condition was considered of sufficient importance nearly a century ago to merit some 30 pages in Politzer's classic textbook. The paucity and mildness of the symptoms, the fact that the great majority of these patients are young children and the lack of careful examination and keen observation on the part of the otologist account for the failure to diagnose this condition more frequently. We now know that the great majority of these cases have an allergic basis and that treatment of the accompanying nasal allergy is often necessary in order to control this condition and to prevent recurrences. The failure to recognize and control secretory otitis media in children is responsible for many of the hearing defects discovered later in life. I believe many of these cases of allergic rhinitis and secretory otitis media would be prevented by proper feeding of young infants. The infant's head should be kept elevated at least 30 degrees from the horizontal when it is fed from a bottle, otherwise it cannot swallow without forcing fluids into the nasopharynx. The frequency of childhood allergies to milk and orange juice may well be due to this insult to the tissues of the nasopharynx.

One of the greatest otologic achievements of the present century has been the development and perfection of the fenestration operation for clinical otosclerosis. This operation already has restored practical hearing for speech to thousands of individuals who otherwise would have gone through life hopelessly hard of hearing. It should be remembered, however, that this operation is purely a mechanical procedure to by-pass the oval window which has become obstructed because of the fixation of the stapes by an overgrowth of bone. The operation will not restore any hearing loss due to nerve degeneration. The operation is definitely an elective procedure, since an individual who will hear well following the operation will also hear well with a hearing aid. Time alone will tell how permanent is the hearing improvement obtained by this operation and whether or not secondary nerve degeneration continues at the same pace as in the unoperated ear. Reports of 10 and 20-year results will be most interesting. Conservative medical treatment of clinical otosclerosis has failed to prove of any value whatsoever up to the present time.

Deafness due to fluid changes and distention of the endolymphatic system occurs in Ménière's disease. Although the deafness simulates a true nerve deafness with poor bone conduction and uniform hearing loss throughout the scale, actual damage and degeneration of the nerve cells may not occur for years. There is increasing evidence that this is primarily a cochlear disturbance and that vestibular involvement and attacks of vertigo may be prevented by prompt and early treatment. The fluctuating degree of deafness with roaring tinnitus, distortion of sounds and hypersensitivity to loud noise makes the diagnosis relatively easy. The hearing loss is reversible in some cases if treated early with peripheral vasodilators or histamine hypodesensitization, although no specific treatment has yet been found to yield uniformly good results. The basic cause of this condition is probably an intrinsic allergy affecting the internal auditory artery and its branches.

Perceptive or nerve deafness due to vascular disorders is definitely on the increase. As the average span of life continues to lengthen, we will see more and more cases of high-tone nerve deafness due to gradual vascular degeneration and an inadequate blood supply to the cochlea. This deafness is irreversible and progressive, and local therapy is of no value. Cardiovascular regulation may retard the progress of the hearing loss.

Cases of traumatic nerve deafness due to explosives or to continuous or repeated exposure to excessive noise have increased tremendously in the past decade largely because of the recent world war. In our modern machine age there are many occupations in which the worker must labor in an environment of noise at a dangerously high level. There is a wide

ation in individual tolerance to excessive noise. It has
my observation that the hearing of an ear with a large
straight external auditory meatus seems much more sus-
ible to damage than one with a small, curved meatus.
strial organizations are becoming more interested in this
lem because of their possible liability to pay disability
pensation for deafness. Some are now requiring audio-
ns of new employees so as to eliminate liability for deaf-
acquired prior to employment. Sound treatment of build-
and insulation of machinery help to lower the noise level.
defenders should be provided when a dangerous noise
l is unavoidable. The general public is becoming more
e conscious and many complaints are being registered
ut unnecessary and excessive city noises. Unless some
sures are taken to reduce the general noise level of our
es and towns, I predict that the acuity of hearing of the
eral public will gradually be lowered before many genera-
s have passed.

ne other type of deafness which is rarely recognized is
ctional or psychogenic deafness. Studies at the army reha-
tation centers during the past war revealed a surprising
idence of nearly 20 per cent of cases of functional deafness
ong the total cases of hearing disorders which they sur-
ed. Audiology clinics are now picking up from 4 to 5 per
t of the cases referred to them. An important finding is
fact that the majority of the cases of functional deafness
e an underlying organic deafness of greater or lesser
ree. The management of these cases is very unsatisfac-
y at present. They need psychiatric aid, but there are too
psychiatrists and many of them are indifferent or unin-
ested in this type of case. There seems to be little doubt
t that many of the so-called miracle cures of deafness fol-
ving the use of some pet remedy have been really the result
a functional alteration in these psychosomatic cases.

Only the very few of us who have had the experience of
earing carbon types of hearing aids with distorted amplifi-
tion, loud friction noises and large, cumbersome batteries
n fully appreciate the improvements which have been made

in the development and perfection of modern hearing aids. These are proving a great boon to nearly a million people who have defective hearing which has not been improved by medical or surgical therapy. I, personally, can hear better and understand more with my present hearing aid than I could 15 years ago, even though my hearing loss for speech has increased from a 50 db average to an 85 db average during the intervening years. Otologists should have a working knowledge of the characteristics of hearing aid amplification and should have an understanding of what hearing aids can and cannot accomplish. For people with a conductive or mixed form of deafness the present hearing aids perform very satisfactorily with uniform amplification throughout the scale. It is much more difficult, however, to provide a satisfactory hearing aid for cases of perceptive or nerve deafness, especially if there be a disproportionate loss of hearing for different parts of the scale. Hearing aids are unsatisfactory and, in some instances, of no value for cases with good hearing for the lower scale, but an abrupt, severe loss of hearing for tones above 1000 cycles. This is the type of hearing loss so commonly found in cases of traumatic deafness and in presbycusis. Fortunately, these people can converse readily with one or two persons in quiet surroundings. The louder the background noise the more difficult does it become to interpret individual voices. Amplification of sounds, including the background noise, does not help the power of interpretation. A small ear trumpet is often more helpful for this group of cases than an electric aid.

For the severely deafened and especially for young children with defective hearing much more help is needed than can be obtained from a hearing aid alone. This is where an audiology department or aural rehabilitation clinic renders valuable service. Speech reading and auditory training are of great help to the severely deafened for whom a hearing aid alone is insufficient to provide the ability to understand readily normal conversational speech. Another valuable service is the preschool training of young children and their parents. Formerly, the deafened or defective child frequently was neglected until it reached the public school age and valuable years of training were lost. These clinics are of great help in analyzing hearing defects and in solving diagnostic problems such as the detection of functional or psychogenic deafness. Their busiest service is hearing aid evaluations and educational training in the proper usage of hearing aids. A well equipped audiology department is expensive and will not be self-supporting, but it provides a most important public service and merits serious consideration in the allotment of public funds and subsidies by foundations and universities.

What is the present situation regarding the clinical management of deafness? It is quite good insofar as reversible forms of deafness are concerned; however, the majority of hard-of-hearing patients who consult us have irreversible forms of deafness which are not amenable to treatment. We fail badly in our management of this large group. For some strange reason many doctors refuse to admit that they cannot improve the hearing of these patients by means of local treatments. Also many false diagnoses are made because of cursory examinations and insufficient study.

Cases of conductive deafness, without suppuration or obvious damage of the conductive apparatus, too frequently are classified as having "chronic, catarrhal otitis media" and are treated with repeated inflations, regardless of whether or not the hearing may improve. The term "chronic, catarrhal otitis media" is a trash pile diagnosis and should be eliminated from our medical terminology. Unless prompt improvement of hearing occurs following two or three inflations, this type of treatment is usually contraindicated. Reports of thickened, retracted and distorted eardrums are often erroneous and the eardrums merely show variations of the normal. Too many cases of otosclerosis are treated year after year with inflations and massage. A pneumatic ear speculum is rarely employed to determine whether or not the eardrums are mobile and the middle ears ventilated. If the patient complains of a lack of hearing improvement, the inflations may be continued anyway to prevent the hearing from getting worse, though irradiation and operative procedures may also be tried. Many tonsils, teeth and nasal septa are sacrificed on general principles to keep the patient satisfied and hopeful of improved hearing.

Cases of mixed deafness and of perceptive or nerve deafness are subjected to a whole gamut of treatments depending on the imagination and ingenuity of the therapist. Irradiations, vitamins and shots in the arm are added to the basic office treatments of nasal sprays and inflations. In the past 30 years the following types of therapy have enjoyed varying degrees of popularity: Subaudible vibratory machines to stimulate the organ of hearing; massage and titilation of the Eustachian tube orifices; irradiation; thyroxine; prostigmine; Jacobsen's solution; various vitamin combinations, most recently large doses of vitamin A; another recent form of therapy is based on the finding of hypercholesterolemia and hyperpyruvemia. Some of these forms of treatment were based on apparently sound, scientific reasoning and were tried out by many of us before it became apparent that they were of little or no value. If vitamin deficiencies were so important in causing nerve types of deafness, it seems very strange that surveys of large groups of evacuees from Japanese concentration camps in the past war failed to show any greater incidence of auditory nerve defects than similar groups of healthy troops, even though the evacuees suffered from extreme malnutrition and avitaminosis. Is it a mere coincidence that new remedies for deafness so frequently reach the headlines of the public press? It should be noted that many specific remedies have been advocated for deafness in general without consideration of the actual type of deafness or its cause. People with irreversible hearing defects can be counted by the million, most of them unwilling to accept the fact that their deafness cannot be cured and eager to try any new form of therapy purported to improve their hearing. They afford a lucrative source of income to unscrupulous therapists.

Why do these various types of treatment attain such popularity and why do we read so many reports of improved hearing following these treatments? The answer lies in the curious psychology of the hard of hearing.

It is a natural reaction for a hard-of-hearing person to have a sense of inadequacy and abnormality and to develop a severe inferiority complex when he has difficulty in conversing with his normal fellow men. In the race for survival of the fittest and in the competition to earn a livelihood, he feels definitely handicapped. The so-called selfishness of the deaf is the result of his failure to make an effort to overcome this handicap. Since he cannot hear well or readily understand what is said to him, he stops listening. Lack of attention and failure to listen make him seem much deafer than he really is. Hearing tests and audiograms are inaccurate since they are subjective tests. If he does not listen attentively he will naturally make a poor score. A hearing aid is repugnant to him as it advertises his infirmity. He wants a miracle, not crutches. If he consults an otologist and is told that his hearing cannot be improved and that he should try a hearing aid he becomes resentful and indignant. He is now ripe for plucking by any therapist who will tell him that his deafness can be cured or helped by treatments. The initial effect of these treatments is often an apparently marked improvement of hearing, noticeable not only to the individual but also to his friends and associates. Audiograms also will show improved scores. This improvement, however, may be purely illusionary. With the hope and expectation of recovering his hearing loss, he becomes alert and attentive. He starts to listen again, which accounts for the apparently improved hearing. When, however, further improvement fails to occur, he soon becomes despondent and ceases to listen, whereupon the initial improvement is lost. The fear of further hearing loss and especially of total deafness may influence him to continue treatments even though he may subconsciously know that he is not being helped by them.

I occupy an almost unique position, since I am a severely deafened otologist. I wasted from five to 10 of what should have been the best years of my life as a selfish, depressed and self-pitying young man. I experienced the wild hopes and bitter disillusionments of futile treatments for my deafness. I not only was harming myself, but I also became a burden to my family and a nuisance to my friends. I was like an ostrich with my head in the sand trying to conceal something which everyone already knew. It was not until I finally accepted my deafness as an unalterable fact and acquired the determination to overcome my handicap that I really began to enjoy life again. I learned to listen not only with my ears but also with my eyes and my brain. Today this is called speech reading and auditory training. I learned to use a hearing aid effectively, though I admit that the early models were very unpleasant with their distorted amplification. One of my most difficult tasks was the re-education of my own speech. With both ears plugged by ear receivers I found it very difficult to modulate my own voice. Even at present I am liable to shout when excited or mumble inaudibly when embarrassed. From my own experience I have learned that the handicap of deafness is about 90 per cent imaginary. When we cease waiting for miracles and are willing to accept crutches we soon discover that the crutches need not be at all burdensome.

It has been only during the past 10 years that I have fully appreciated what a tremendous asset my deafness has been to me in the practice of otology. There is a feeling of comradeship and understanding between a hard-of-hearing patient and his hard-of-hearing physician which could not occur if the physician's hearing were normal. Patients will accept advice and opinions from me which I know they would not accept from a physician with normal hearing.

The otologist has a definite obligation to his hard-of-hearing patients. If medical and surgical therapy are ineffective in improving the hearing, psychologic therapy is still available and badly needed. Fifteen to 30 minutes spent in a frank discussion of his problems will often give the patient greater help than months or years of futile treatments.

In closing I offer a few final words of advice on the management of patients with irreversible hearing loss.

Discuss their problems with them frankly.

Tell them the nature of their deafness and why they cannot be helped by medical or surgical treatment.

Gain their confidence by telling them the score and what they can do to balance the score.

Be positive and firm with your advice but give them encouragement.

Do not give them sympathy. They crave sympathy but already pity themselves too much.

Do not tell a patient his hearing will get worse. Hedge, if you must, by saying that adjustments, training and hearing aids will more than counterbalance any further hearing loss.

Never tell a patient that he will become deaf. The greatest phobia the hard of hearing experience is the fear of total deafness. Reassurance on this point alone frequently will be of more benefit to the patient than all the medical and surgical therapy we can offer.

It takes time and effort to convince hard-of-hearing people that they must accept their handicap as an actuality before they can be guided onto the path leading to necessary readjustments and rehabilitation. If we consider ourselves to be real otologists we will gladly expend this time and effort in their behalf.

121 University Place.

PRIMARY TUMORS OF THE NASAL SEPTUM.*

F. A. SOOY, M.D.,
San Francisco, Calif.

Primary tumors of the nasal septum are not common. The various types encountered in this location and the problems peculiar to their treatment are discussed. Eleven patients with tumors of the nasal septum are reported.

Theoretically, a tumor arising from any of the histological elements of the septum could occur in this area; however, a review of the literature reveals certain types to predominate.

Hemangiomas: A variety of tumors of blood vessel origin have been reported, and these are classified according to the predominant vascular element into cavernous and simple types. The common location is Kiesselbach's area (see Fig. 1).

Cases have been reported by Callison,[1] Tilley,[2] Rosenthal,[3] Dabney,[4] Scuderi,[5] Franchini,[6] Klemptner,[7] Maekawa,[8] Weidlein,[39] and Salinger.[40] Salvadore[9] reported two cases, one a cavernous angioma and the other a hemangioendothelioma. Weidlein[10] reported three cases, one with a question of early malignant change.

Clinically these are characterized by repeated nasal bleeding and varying degrees of obstruction, depending upon the tumor size. Examination reveals a reddish-brown polypoid mass attached to the septum, usually by a narrow base. Manipulation frequently results in vigorous bleeding (Case 4). The usual anterior location of these lesions suggests that digital trauma may be an etiological factor (Case 8).

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*Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1950.

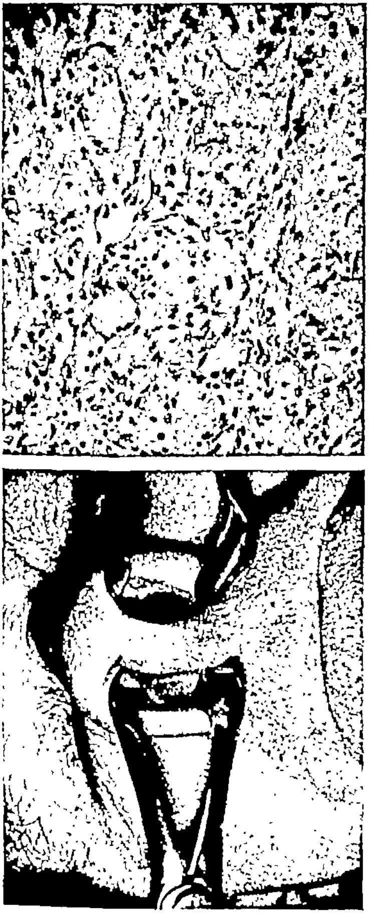


Fig. 1. Black and white print from the kodachrome original of Case 4 showing the pedunculated hemangioma in Kiesselbach's area.

Local excision with cauterization of the base is usually adequate for the small lesions. The more extensive tumors are best treated by preliminary radiation[10] to reduce the vascularity followed by surgical excision.

Endothelioma: Eggston[10] states that endotheliomas in the nose are presumably often classified as "mixed tumors of salivary type." He feels many endotheliomas might be more accurately labeled mixed tumors, and that they should be considsidered to be of borderline malignancy.

Higushi and Shirawa[11] reported a hemangioendothelioma of the septum, Zarahune[12] a myxoendothelioma, and Gon[13] a lymphendothelioma of the vestibule. In appearance and symptoms these tumors are similar to those outlined for hemangiomas (Case 9). Cautery excision is usually adequate.

Mixed Tumors: These are smooth, encapsulated, slow growing tumors composed of several tissue elements. There is a marked tendency to recur after incomplete excision. Cauterization is a useful adjunct.

Stevenson,[14] Weidlein[15] and Ersner[16] have each reported a case. Ersner's was successfully removed by an external incision. Nasal obstruction and external deformity were present. Vermeuw and Goormaghtgh[17] report a cylindroma of the septum.

Adenoma: Eggston[10] states that adenomas in the nose are usually on the septum or turbinates. They appear as red sessile or pedunculated vascular masses and are amenable to wide local excision.

Fibroma: A pedunculated fibroma of the septum was described by Simpson,[18] who states that granulomas, angiomas and fibromas are usually in the anterior half of the septum while myxomas are located in the middle or posterior third. Silva[19] reported a large, slow growing fibroma of the septum. In both cases removal was accomplished surgically.

Grossly these lesions are pale, firm, mucosal-covered, pedunculated lesions which enlarge very slowly. Local excision is the treatment of choice.

Neurilemmoma: Bogdasarian and Stout[20] describe a polypoid nerve sheath tumor which was snared from the septum and the margins cauterized. They were unable to locate any similar case in the literature.

The mass is described as a reddish-pink, ovoid, firm, nontender mass attached to the left side of the septum in Kiesselbach's area. Case 11 is a similar tumor with more extensive involvement of the septum. Local excision without cauterization was effective.

Dermoid Cyst: Juers[21] reports one case removed intranasally.

Plasma Cell Tumor: Blumenfeld[22] in his review of plasma cell tumors of the nose and nasopharynx includes one case involving the nasal septum reported in 1925, by Facchini and Scalas.

Clinically the mass is soft, gray and friable and bleeds easily on manipulation. Local bone involvement occurs. Radical local excision and cauterization is indicated.

Chondroma: Howarth[23] reports one case of chondroma involving the anterior septum which excised surgically. He found two other cases in the literature.

The gross appearance is similar to that of other chondromas and the symptoms are those of obstruction and external deformity. Local excision if complete is all that is required.

Chondrosarcoma: Owen[24] describes a patient with a red, smooth mass arising from the nasal septum with occlusion of both posterior nares. This was removed and a microscopic diagnosis of chondroma was made. The lesion recurred, however, and further examination of the tissue subsequently removed suggests a chondrosarcoma. Radical local excision followed by cauterization or radiation is indicated.

Giant Cell Tumor: Bjuggren[25] reports a patient with a giant cell tumor of the right nasal cavity; the origin, however, was from the turbinate rather than the septum. Case 3 represents a true giant cell tumor of the nasal septum. The external appearance was that of a simple, firm, fibrous polyp of the anterior portion of the nasal septum. No ulceration was present (see Fig. 2). Local excision should include the underlying cartilage.

Papilloma: Geschickter[26] feels that benign epithelial tumors of the nose are rarely reported because they are slow growing and relatively asymptomatic. Kramer and Som[27] classify papillary growths in the nose as follows:

1. Mucus polyps.
2. Cutaneous warts. These are usually located on the nasal septum or vestibule.
3. Papillary carcinoma or malignant papilloma.
4. True papilloma or papillomata dura. These are of two types, one consisting of a cauliflower-like growth with a narrow or wide base (see Fig. 3), and the other composed of diffuse small nodules.

In reviewing the literature to 1856, they have listed five instances where the papilloma primarily involved the septum.

There is a marked tendency for local recurrence. Klemptner[7] reports a case with diffuse involvement of the nostril which he removed five times in five months. Nasal obstruction is the chief symptom and malignant degeneration can occur. Wide local excision with extensive cauterization of the margins is indicated.

Epidermoid Carcinoma (Papillary) or Malignant Papilloma: Kramer and Som[27] report a papilloma which underwent malignant degeneration and Case 2 shows a similar process in the nasal septum (see Fig. 4).

Clinically these are extremely vascular, friable, reddish-gray tumors which bleed frequently and profusely. The bleeding may be noticed long before the obstruction becomes a problem. Wide local cautery excision is indicated, followed by local radiation in large lesions. A neck dissection need not be routinely carried out. Hemorrhage is a problem.

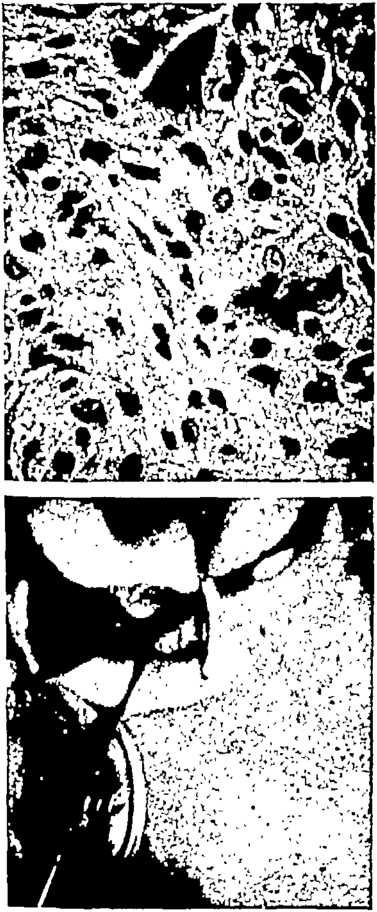


Fig. 2. Black and white print from the kodachrome original on Case 3 showing a pedunculated giant cell tumor in Kiesselbach's area on the right side of the nose.

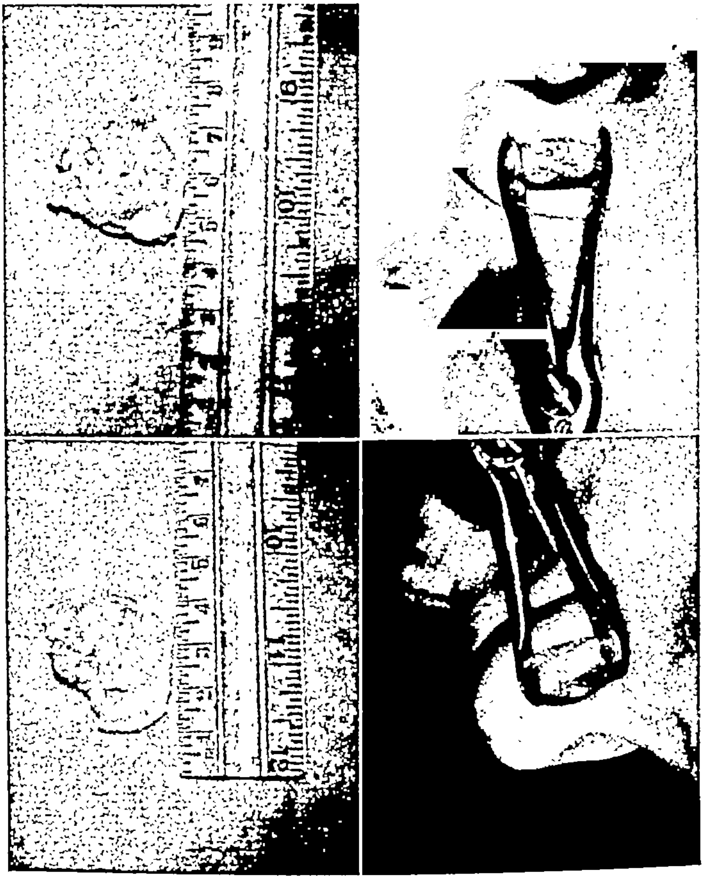


Fig. 3. Black and white print from the kodachrome original of Case 10 showing an extensive, warty, squamous papilloma which involves both sides of the cartilaginous and bony septum. Both sides of the resected septum are shown.

Lymphoepithelioma: This lesion is usually confined to the nasopharynx; however, one case involving only the posterior nasal septum is reported (see Fig. 5). Treatment has included external radiation, radium, surgical excision and electrocoagulation.

Malignant Melanoma (Melanosarcoma): Smith[36] warns that malignant melanoma must be considered in the differential diagnosis of papillomas of the anterior nasal cavity.

The origin of malignant melanomas within the nasal cavity is not clear. They may arise from misplaced cutaneous cell

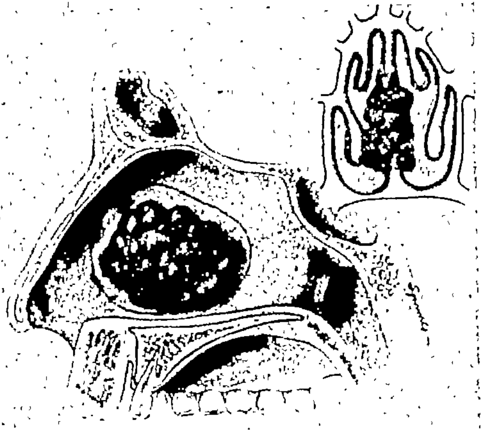


Fig. 4. Semischematic drawing of a papillary epidermoid carcinoma primarily in the septum with a "kissing" metastasis of the right inferior turbinate, Case 2.

rests and are also seen in the rectum, which is another mucocutaneous junction. The growth of these lesions is slow prior to metastasis and they are amenable to surgical excision with or without radiation. Kaplan[28] states that the common location of these tumors is either on the septum or lateral nasal wall.

The gross appearance of the lesion is a mushy, black, polypoid mass which bleeds readily on manipulation. The chief symptoms are obstruction to breathing and bleeding. Metastasis occurs in approximately 50 per cent of these lesions.

Cases have been reported by Lewy,[29] McKensie,[30] Smuts,[31] Munroe,[32] Margarat, *et al.*,[33] Fujita,[34] and Brandao.[35] Recurrence among the reported cases is frequent. Radical local excision with appropriate neck dissection followed by radiation seems to offer the best hope of arresting this disease.

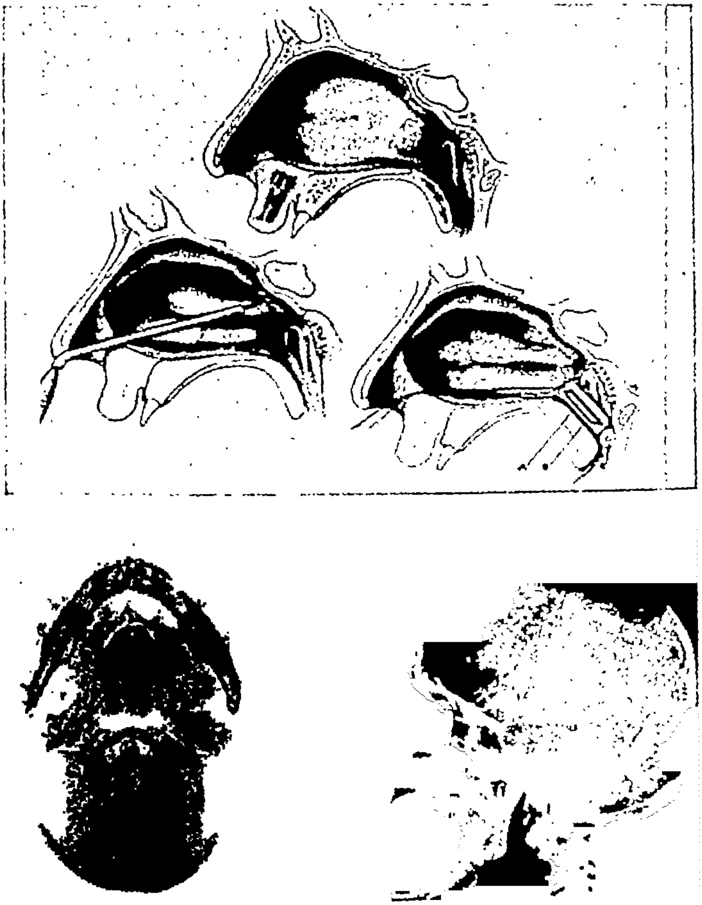


Fig. 5. Semischematic drawing showing a small lymphoepithelioma involving the bony posterior septum, Case 1. The septal resection was carried out with punch forceps and the base electrocoagulated as shown with a special nasal tip. (A) Dental wax block containing four 10 mg. radium needles. (B) Curved lead shield used to protect the underlying palate. (C) Gauze postnasal plug. Radiograph shows the lead shield and radium in situ.

Hypernephroma: Fanoe[37] reports a hypernephroma of the nasal septum in a patient who previously had a kidney removed for this condition.

Sarcoid: Fletcher[38] describes four patients with sarcoid of the nose. Three of these had primary septal involvement.

They were characterized by small, reddish, granular nodules on the septum. He reports that surgical excision was successful in two cases. Electrocoagulation did not seem too effective.

Tuberculosis: Videgain[41] reports a case of tuberculous granuloma of the septum. This as well as syphilis must always be considered in the investigation of any septal mass.

Treatment: In many cases reviewed from the literature, and in Case 3 reported here, inadequate removal of the original lesion resulted in recurrence. This necessitates subsequent radical resection which might not otherwise have been necessary. One cannot stress too strongly the importance of the initial treatment. This procedure should always be more extensive than would first seem indicated.

The following procedures have been adopted as a result of the preceding study:

1. Small posterior polypoid lesions are resected locally and the base cauterized.

2. Small, discrete, anterior lesions are not biopsied. The surrounding mucosa and attached cartilage are removed under local anesthesia, *leaving the mucosa on the opposite side intact* (see Fig. 6). Vaseline gauze packing is placed over the defect for 24 to 48 hours and no cauterization is used. Complete epithelization occurs over the intact mucosa of the opposite side in four to six weeks. Vaseline is used to prevent crusting and there is no disability during this period. More extensive lesions are first biopsied for diagnostic purposes and this is checked against the cytological smear findings.[42]

3. Large, benign, mucosal-covered lesions are resected intranasally with an adequate margin and the edges of the septum are cauterized (see Fig. 7). General anesthesia is preferred because of the discomfort incident to adequate coagulation. When the lesion involves the bony septum the cartilaginous portion is excised *en bloc* and that attached to the bony portion is removed with punch forceps. There is sequestration of devitalized bone fragments from the margin during the healing process. Epithelization is complete in six to eight weeks.

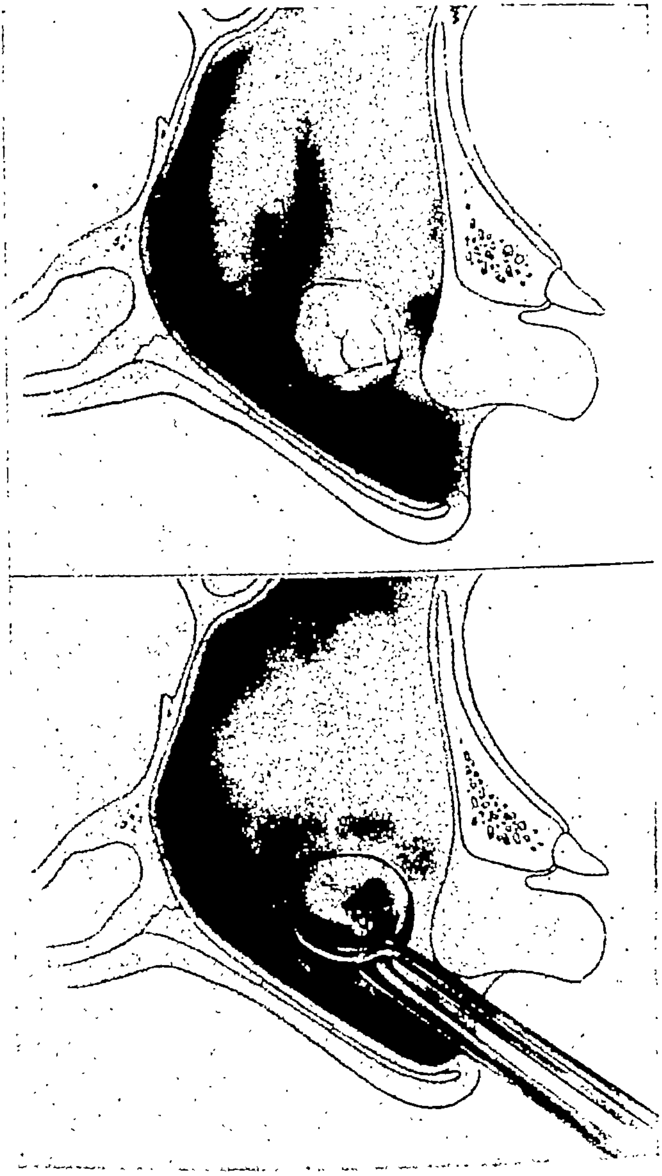


Fig. 6. Semischematic drawing showing the submucous method of excising small lesions with the underlying cartilage, Case 10.

4. Large, fungating, ulcerated, malignant lesions are treated in a similar manner except that the maximum margin is

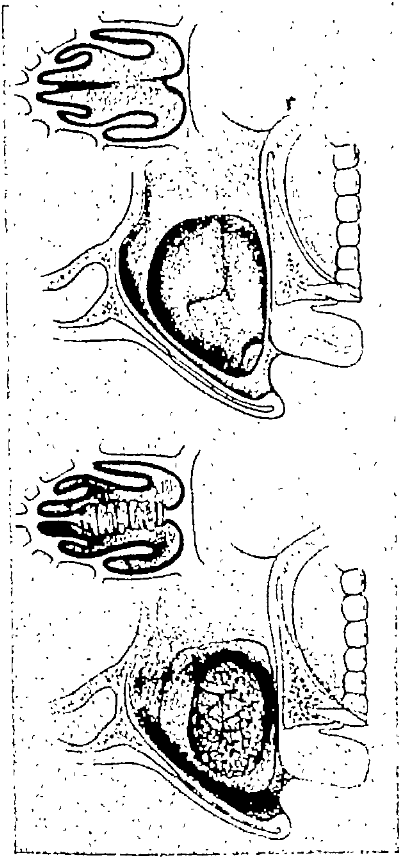


Fig. 7. Semischematic drawing showing the initial lesion and final septal resection for a squamous papilloma. Case 5.

obtained and the subsequent fulguration is very extensive. The period of sequestration and epithelization is consequently prolonged.

5. Posterior malignant lesions are resected intranasally with the entire posterior septum and most of the anterior part (see Fig. 5). The margins are thoroughly fulgurated. The extensive septal resection is carried out in order to facilitate subsequent observation for evidence of recurrence.

The patient is always advised that nasal crusting may be a problem following a radical septal resection. This is controlled by vaseline locally to the anterior naris and steam inhalations during cold weather. Nasal douching has not been necessary. In none of the cases was any postoperative external nasal deformity present.

Recurrences (see Fig. 8) are treated by recauterization, followed by intranasal radium radiation. The septal resection facilitates this procedure (see Fig. 11). Careful advance planning is important so as to have the radium needle attached to a retainer which will remain firmly in the correct position and deliver uniform radiation (see Fig. 5A). The uninvolved area is shielded with a lead plate (see Fig. 5B).

In February, 1948, Morrison and Low-Beer[13] developed a practical method of using Radioactive Cobalt 60 as a radium substitute in the treatment of nasal tumors (see Figs. 9 and 10). This is being used and the results are promising. The half life of Radioactive Cobalt 60 is 5.3 years.

The Cytological Smear Technique as described by Morrison, *et al.*,[12] has been most useful in finding early recurrences (Case 1) and as a simple method of evaluating radiation cellular change (see Fig. 11). They report 138 cases examined by this method and in no instance was a false positive smear obtained. Two cases with negative smears in the presence of a positive biopsy were explained by the absence of involvement or ulceration of the surface epithelium.

Malignant cells may be found in smears obtained from the surface of an ulcerated, malignant epithelial lesion of the nose, throat or larynx, no matter how small. The smear technique is now routinely used in all malignant tumors of the nose and throat. It is most useful in the evaluation of the effect of treatment.

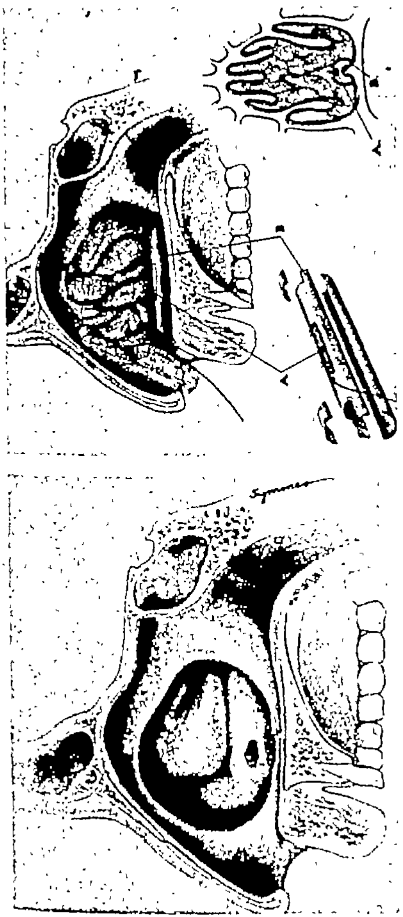


Fig. 8. Semischematic drawing showing small local recurrences in the inferior turbinate and base of the septum. The radium was fixed to a segment of rubber tubing and inserted following recauterization of the lesions. Gauze packing is used for fixation. Case 2.

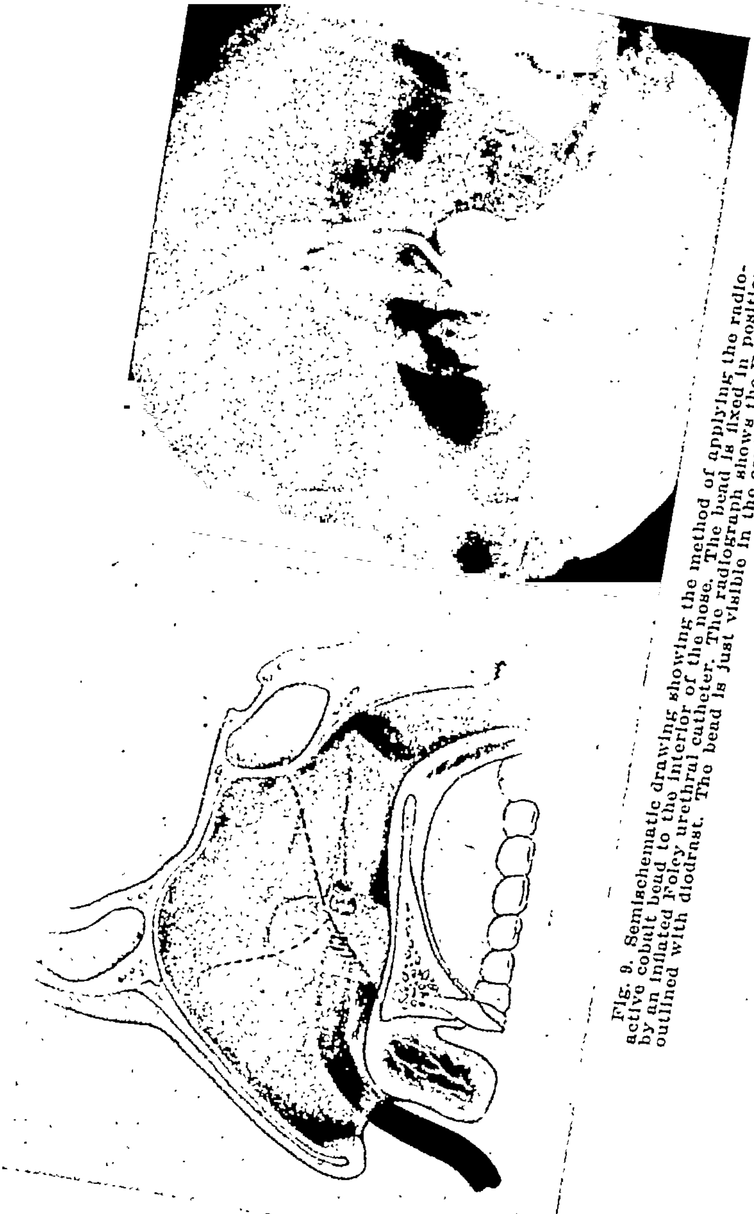


Fig. 9. Semischematic drawing showing the method of applying the radioactive cobalt bead to the interior of the nose. The bead is fixed in position by an inflated Foley urethral catheter. The radiograph shows the Foley bag outlined with diodrast. The bead is just visible in the center of this.

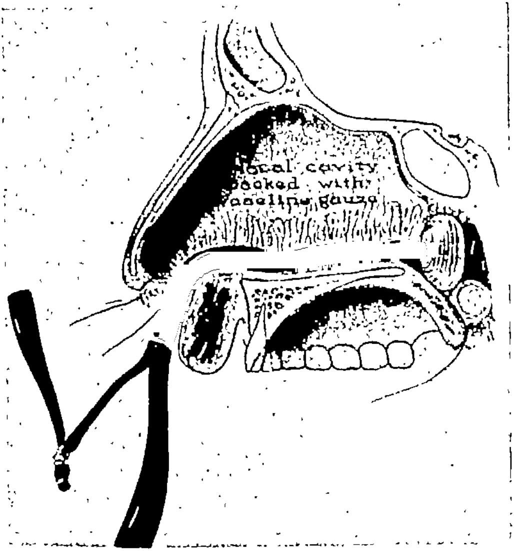
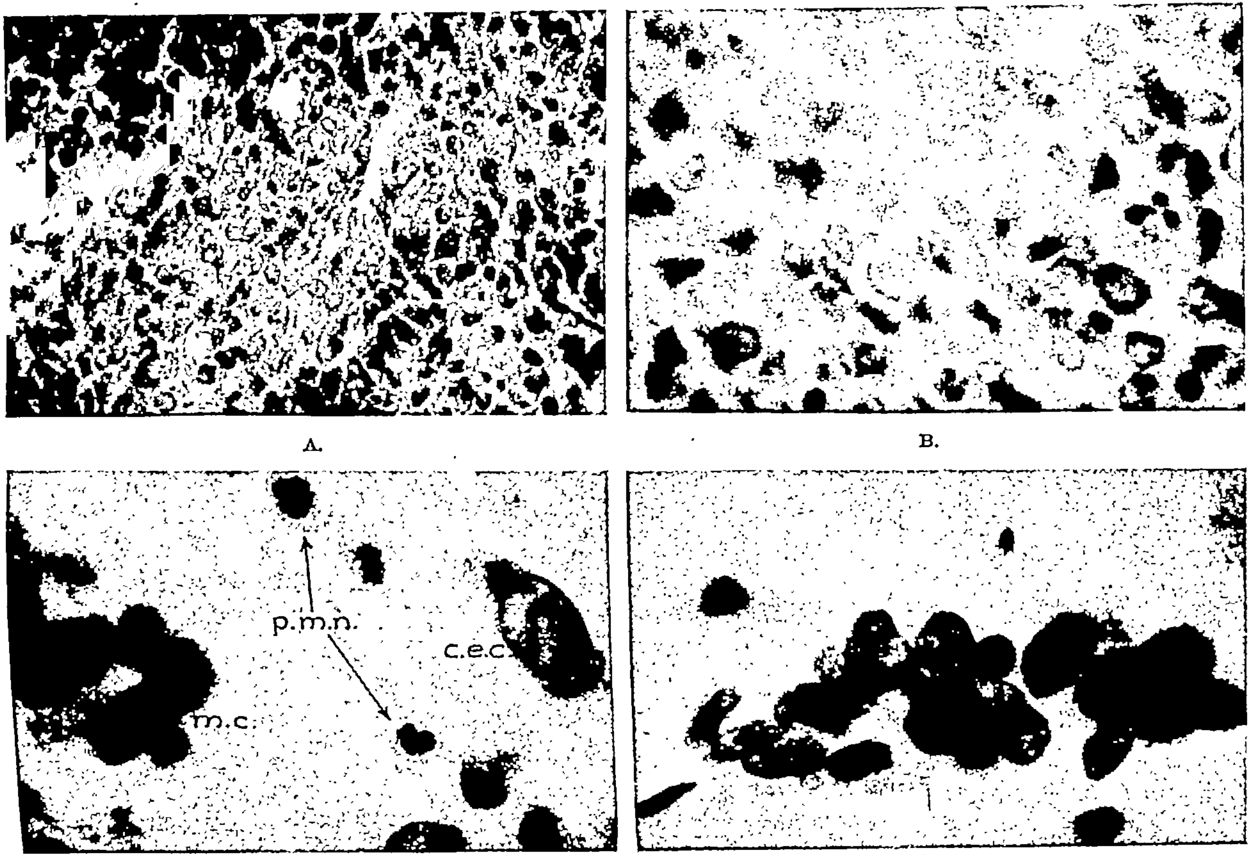


Fig. 10. Semischematic drawing showing the method of treating nasopharyngeal tumors with radioactive cobalt.

Epithelial cells as seen on the smear undergo the following progressive radiation changes:

1. Swelling.
2. Foamy cytoplasm.
3. Nuclear swelling.
4. Granulation of the chromatin.
5. Fragmentation of the cell.


A.

B.

Malignant cells differ from the normal cells by virtue of:

1. Irregular size and shape.
2. Loss of cytoplasm.
3. Coarse patchy chromatin.
4. Thick nuclear membrane.
5. Formation of multinucleated cells with irregularly sized nuclei.

We have not employed the more radical external operations of Mouré and Denker for primary septal tumors. These approaches have been successfully used by others particularly for larger tumors.

Carotid ligation was not found to be necessary even in the largest and most vascular tumor of the series. In one instance (Case 2) packing was necessary to control the massive hemorrhage before electrocoagulation could be completed.

SUMMARY.

1. Primary septal tumors are uncommon.
2. The literature is reviewed and the clinical characteristics outlined.
3. The importance of sufficiently radical initial surgical resection is stressed.
4. An appropriate intranasal surgical procedure for each group is outlined and illustrated.
5. Methods of supplementary intranasal radiation are described and a new method is presented.
6. The cytological smear technique has proven of value in diagnosis and treatment.

7. Eleven illustrative cases are reported. These include:

1. Lymphoepithelioma.
2. Papillary epidermoid carcinoma.
3. Giant cell tumor.
4. Hemangioma.
5. Papilloma.
6. Squamous carcinoma.
7. Inflammatory polyp.
8. Granuloma.
9. Hemangioendothelioma.
10. Squamous cell papilloma.
11. Glioma.

CASE REPORTS.

Case 1: E. M. H., white male, age 59; U. 121770. This 59-year-old male druggist was first seen at the University of California Hospital, Out-Patient Department, on Aug. 12, 1945. A letter stated that in December, 1942, a submucous resection was carried out and a large polyp was removed from the right side of the nose in the region of the right middle meatus. Following this surgical procedure he complained of persistent pain, postnasal discharge and bleeding from the right side of his nose.

Biopsy was taken from this area on June 9, 1943, and this was reported as follows: "The tumor consists of a large mass of cells supported by very small amounts of stroma. These cells are pale staining and have very poor outlines. These are fairly small and have round central granular pale staining nuclei. Intermingled with these tumor cells are found inflammatory cells, lymphocytes and polymorphonuclear series. Mitotic figures are fairly numerous. There is no cellular pattern. *Diagnosis:* Anaplastic carcinoma of the nose, grade 4 (so-called lymphoepithelioma group)."

The patient was given four intranasal radium applications in October, 1943, consisting of contact radium applicators of 50 mg. with 1 mm. platinum filter. A total of 600 mg. hours of contact therapy was given in a two weeks period.

Examination on Oct. 12, 1945, two years later, reveals a small amount of pale pink tissue between the inferior and middle turbinates on the left side posteriorly. No bleeding was seen. There was also a small knob of tissue posteriorly in the left choana at the posterior border of the septum. The remainder of the ear, nose and throat examination was normal. General medical examination was normal except for incipient diabetes mellitus and some arthritis of the lumbar spine. The patient continued to complain of pain, headache and nasal discharge until March 10, 1948, at which time direct nasopharyngoscopy showed a pea-sized, rather granular vascular appearing swelling of the left side of the nasal septum. It was felt that this possibly represented a recurrence and again a biopsy was taken. Microscopic diagnosis of this tissue revealed transitional cell carcinoma of the nasopharynx.

In July of 1947 the patient had received a total of 1,250 r. to a 3 x 5 cm. field over the left side of the nose for a superficial skin condition. Because of this and his previous radiation therapy it was felt that further radiation at this time was inadvisable. He was, therefore, admitted to the University of California Hospital on May 26, 1948, and on the following day was taken to surgery, where under avertin anesthesia supplemented by intratracheal nitrous oxide and oxygen, careful exploration of the nose revealed a raw, ulcerated area on the extreme posterior margin of the nasal septum. This measures approximately 0.5 x 1 cm. size. A previous submucous resection had been done and was well healed except for a 2 x 4 mm. anterior-inferior perforation of the septum just above the vomer process. By means of a sharp knife the entire septum was excised from a point 1.5 cm. posterior to the anterior border of the cartilaginous septum. The bony part of the septum remaining was removed by means of sharp rongeurs and the vomer ridge was taken down flush with the palate by means of a mallet and gouge. Considerable bleeding was encountered but this was controlled by means of packing. At the close of the procedure the entire posterior portion of the nasal septum was extensively coagulated by means of a Bovie unit. This fulguration included the anterior base of the sphenoid, and the base of the bony septum inferiorly. No evidence of tumor was found in the portions of the septum which were resected. Smears taken from the lesion prior to surgery and stained by the Papanicolaou stain showed evidence of malignancy in the nasal secretions.

He was followed in the outpatient department and continued to complain of pain and headaches. Re-examination of his Papanicolaou smears on July 28, 1948, showed malignant cells to be still present in the nasal secretions.

X-rays of the base of the skull showed no evidence of malignant involvement of this area.

The patient was again admitted to the University of California Hospital on Aug. 13, 1949, and taken to surgery on the following day, at which time he was anesthetized by means of avertin and intratracheal nitrous oxide and oxygen anesthesia. Inspection of the nasopharynx and vault and base of the nasal septum failed to reveal any evidence of gross malignancy. Biopsy specimens were taken from the vault, the base of the septum and the nasopharynx, after which a very thorough cauterization was carried out in Rosenmüller's fossa and over the vault in the nasopharynx. Similar cauterization was carried out at the base of the nasal septum and the posterior portion of the hard palate. No bleeding was encountered and the patient was returned to the ward in good condition.

Biopsy report of the specimens obtained from this procedure showed nasopharyngeal mucosa with acute and chronic inflammation and granulation tissue. There was no evidence of malignancy.

The patient continued to complain of headache, pain and bleeding from his nose and smears taken from a roughened area at the vault of the nasopharynx continued to reveal positive malignant cells by the Papanicolaou technique.

The patient was readmitted to the University of California Hospital on Oct. 13, 1948, and on the following day he was taken to surgery and anesthetized by means of cyclopropane and ether anesthesia. The nasopharynx was exposed by means of a tonsil gag into which a hard, wax plaque containing four 10 mg. radium needles in a rectangular fashion was inserted into the nasopharynx so as to obtain uniform radiation over the vault of the nasopharynx and the superior margin of the nasal septum. The soft and hard palate were protected from radiation by means of a lead shield which was fixed in position by means of anterior linen strings which were tied to an anterior nasal bolster. The lead plate and radium pack were held in position by anterior and posterior gauze packing which was allowed to remain in place a total of 26 hours, which amounts to 1,040 mg. hours at 1 cm. or is the equivalent of 4,900 r. equivalents.

Following the application of the radium the patient improved markedly, his pain disappeared and except for some drying and crusting in the nasopharynx he was without symptoms. His Papanicolaou smears became consistently negative and have remained so to the present time, now nearly one year after his last treatment.

Case 2: M. D. C., white female, age 64. The patient was admitted to the University of California Hospital on Oct. 16, 1946, complaining of a progressive nasal obstruction associated with postnasal discharge from the right nostril of one year's duration. She had had two biopsies prior to admission, followed by extensive and profuse bleeding from the right side. There was no obstruction to breathing on the left side. There were no other significant findings in the history.

Examination of the nose revealed a soft friable mass involving the right side of the nasal septum and extending submucosally beneath the left side of the nasal septum. This mass occludes the entire nasal fossa on the right side from the vestibule anteriorly to the floor inferiorly and does not involve the posterior choana. The color is a pale pink hue. By means of nasopharyngoscopy the mass on the right side could be seen to extend almost to the posterior naris on this side but did not involve it by actual extension. No cervical glands were present on either side. The remainder of the ear, nose and throat examination and general physical examination were not remarkable.

On Oct. 17, 1946, the patient was taken to surgery, and under local anesthesia the entire septum including the tumor mass was resected, care being exercised to leave sufficient septum anteriorly for support of the nose, and a small amount of the posterior margins of the bony septum was found to be uninvolved and this was left intact. It was planned to carry out an extensive cauterization of the margins of the defect at this time; however, the bleeding was so profuse as to preclude the possibility of effective coagulation. Consequently the area was packed by means of anterior-posterior pack. The patient was returned to the ward in good condition. She was given a 500 cc. whole blood transfusion the afternoon of the day of surgery and three days later under general anesthesia with intratracheal intubation the nasal packing anteriorly and posteriorly was removed cautiously and the underlying tissue cauterized as the packing was withdrawn. This way a very thorough electrocoagulation was carried out on all the margins of the remaining nasal septum and of the adjacent right inferior turbinate where a small kissing lesion was found to be present following removal of the main mass. The area was again packed and the patient returned to the ward in good condition. The packing was slowly removed on the second and third postoperative days, and her postoperative convalescence was uneventful.

She was discharged on Oct. 23, 1946, with instructions to return weekly for observation. Her laboratory examination, including routine blood, urine and serology, was negative.

Pathological Report: Gross Description: The specimen consists of eight irregular pieces of tissue; five are pale pink with a cauliflower appearance, ranging in size from 1.5 x 1 cm. to 2 x 1.5 cm. Two of similar size are flat with smooth, grayish-pink surface. The eighth piece is a piece of cartilage measuring 0.2 x 0.3 cm.

Microscopic: The cauliflower masses show stratified, apparently squamous epithelium that is hyperplastic, forming a papillary projection with thin connective tissue stroma. The cells are hyperchromatic and pleomorphic with marked variation of nuclear size and frequent mitoses. The remaining tissue consists of respiratory epithelium overlying a connective tissue containing many mucous glands. The cartilage is normal, but contiguous to it are masses of large epithelial cells with pale, spongy cytoplasm and irregularly sized nuclei showing occasional mitosis.

Diagnosis: Papillary epidermoid carcinoma.

Examination one month later revealed evidence of a small local recurrence on the right inferior turbinate. Consequently on Nov. 21, 1946, she was readmitted to the University of California Hospital and under local anesthesia a very extensive electrocoagulation was carried out, particularly over the right inferior turbinate, and again the margins of the nasal septum resection were cauterized with electrocoagulation. Biopsies were taken prior to coagulation. Pathological report of these specimens reveals recurrent carcinoma of the squamous cell type in the nasal septum. This carcinoma was evident both in the specimen removed from the right inferior turbinate and that removed from the base of the septum. The patient's course following this coagulation was uneventful for approximately nine months, at which time she showed evidence of a very small recurrence at the base of the septum in the midportion of the vomer. She was therefore readmitted to the University of California Hospital on Aug. 29, 1947, at which time a 25 mg. radium needle was attached to a half section of rubber tubing which was so cut as to slip into the septal defect and fix the radium over the local recurrence. This was held in position by a packing. She was given a dose of 250 mg. hours, which is the equivalent of 6,250 r. Following the removal of the radium her subsequent course has been entirely uneventful. When she was seen in May of 1949 there was no evidence of recurrence two years and six months after her original diagnosis was established.

Case 3: C. N. S., white male, age 52 years; U. 43596. This unemployed male was first seen in the ear, nose and throat clinic on June 20, 1939, complaining of frequent bleeding from the right nostril. A small polyp was seen on the right side of his nasal septum in Kiesselbach's area and this was the lesion which was producing the bleeding. It was cauterized with chromium trioxide, and he had no more trouble with his nose until he presented himself again in the ear, nose and throat clinic on July 29, 1947, eight years later, complaining of consistent nasal obstruction on the right side of five months' duration. The nasal obstruction is almost complete and constant, associated with crusting in the right nostril.

Examination revealed a 1 cm. polypoid mass arising from Kiesselbach's area on the right side of the nasal septum which was ulcerated on the anterior surface. The mucosa otherwise appeared normal. The area was photographed and the lesion was excised locally with cautery to the underlying base.

Microscopic examination of the excised tissue showed a superficial layer of stratified squamous epithelium and small areas of erosion covered by coagulated fibrin and necrotic cells. The epithelium is thick and has papillae which are narrow and deep. There is no invasion or evidence of malignant change. The underlying connective tissue contains a condensation of cellular connective tissue elements. Large fibroblastic cells of varying size and shape are present between strands of connective tissue. Giant cells with eosinophilic cytoplasm and five to 10 centrally placed small oval nuclei are interspersed throughout the tumor and are most numerous adjacent to the osteoid tissue. Numerous small, vascular channels are present and are prominent in some places. A few small accumulations of brown pigment granules are noted within the growth. Mitoses were not seen.

Diagnosis: Giant cell tumor of the nasal septum.

X-rays of the skull with special attention to the nasal septum failed to reveal any evidence of disease involving the nasal septum or skull.

Because of the diagnosis of giant cell tumor of the nasal septum and gross evidence of involvement of the cartilage of the septum it was felt advisable to resect the nasal septum beneath the involved area. The patient was admitted to the University of California Hospital on Aug. 29, 1947, and was taken to surgery on the following day, at which time a circular area of cartilaginous septum containing the mucosa on both sides was removed over the area as large as a five-cent piece. This left a large central septal perforation but this was felt preferable to a recurrence of his tumor. The area was packed with vaseline gauze packing, which was removed on the following day, and the patient returned home.

Microscopic examination of the excised septum revealed scattered throughout the mass numerous round or oval giant cells with large amounts of basophilic cytoplasm and three to 12 or 15 centrally placed vesicular nuclei which frequently have a prominent nucleolus. Several small spicules of osteoid tissue and bone are noted adjacent to the attachment of the tumor. The surface is covered by stratified squamous epithelium except for an eroded area where it is replaced by a fibrinopurulent exudate. Diagnosis was giant cell tumor of the nasal septum.

The patient was last examined on May 26, 1948, at which time there was no evidence of recurrence and such crusting mass as was present was controlled by the use of vaseline locally. This was nine months following his septal resection and there was no evidence of recurrence.

Case 4: White female, age 30. This physician's wife was first seen in the office on Aug. 6, 1949, with a history of having had a polyp removed from the right side of her nose six weeks previously. She has had recurrent nose bleeds for one year. This bleeding has continued following the removal of the "polyp."

Examination of the nose revealed a 1.5 cm. circular ulcer in Kiesselbach's area on the right side of the nasal septum with cartilage exposed in the central portion. The patient was advised to use vaseline locally to promote healing and inquiry revealed that a 0.5 cm. polypoid, pedunculated, reddish-purple mass had been snared from Kiesselbach's area on the right side six weeks previously. The base was cauterized with chromic acid.

The pathological *report was as follows:* "The *section is composed of* a mucoid stroma containing numerous large vascular spaces. In one area there are numerous leucocyte collections with necrosis of tissue. If these had eroded into a vessel considerable bleeding could have occurred. *Diagnosis:* Hemangioma."

Subsequent observations on Aug. 12, 19 and 31, 1949, and Sept. 13, 1949, revealed rapid and uneventful healing. The patient is well at the present time.

Case 3: E. P. R., white male, age 48; U. 133351. The patient was admitted to the University of California Hospital complaining of a tumor in the septum of his nose of two and one-half years' duration. He first noticed a small wart in his left nostril two and one-half years ago. He felt that this became infected while working at his occupation as a paint sprayer. He had received a variety of treatments to the area locally, including 300 r. of X-ray therapy and repeated local cauterization by means of a common burning glass using sunlight; all of these without any evidence of regression in size of the tumor mass. The remainder of the history is noncontributory except for an attack of pneumonia in 1919 which resolved very slowly. Question of tuberculosis was raised at this time but could never be confirmed.

Physical examination was normal except for his nose, which showed a large, irregular, warty growth protruding from both sides of the nasal septum anteriorly and extending almost to the bridge of the nose superiorly and the floor of the nose inferiorly. The mass extended approximately 2.5 to 3 cm. posteriorly and involved part of the cartilaginous and bony nasal septum. Both nares were completely occluded by the growth. The superficial surface was whitish in color and warty in consistency. A metal probe could be easily passed through the substance of the wart from one nostril to the other, showing almost complete destruction of the cartilaginous septum. There was no involvement of the nasopharynx or roof the mouth. A biopsy report from this mass was reported by another pathologist as showing an adult, warty growth with marked superficial keratinization and epithelial pearl formation. The microscopic diagnosis was acanthoma. The laboratory work in the hospital was within normal limits.

On Oct. 4, 1946, the patient was taken to surgery and under local anesthesia the entire cartilaginous septum and the anterior half of the bony septum was excised without excessive bleeding. A margin of 1 cm. of good tissue was obtained on all sides of the tumor mass. This necessitated leaving only a 0.5 cm. cutaneous band of tissue anteriorly in the nasal septum; however, it was sufficient to give a normal external appearance. The edges of the excised area were cauterized extensively by means of electrocoagulation and no packing was needed. The patient was discharged on Oct. 6, 1946, after an uneventful hospital stay.

The pathological report of the excised tissue is as follows:

Gross Description: The specimen consists of a resection of the cartilaginous nasal septum. The septum is covered on either side by a thick, yellow mucous membrane approximately 0.2 cm. in thickness and in one extremity gives way to a skin containing multiple hairs. The greatest approximate dimensions of this specimen is 2.5 x 1.7 x 1.3 cm. The cartilaginous septum is 0.1 cm. in thickness on either side of the nasal septum and infiltrating into it is a large grayish-white papillomatous lesion which has infiltrated and destroyed the cartilage of the nasal septum. This specimen has three grossly adequate margins present along one edge of the septum.

Microscopic: The sections show the nasal cartilaginous septum to be eroded away by connective tissue showing marked chronic inflammatory changes. Replacing the cartilage are long cords and columns of stratified squamous epithelial cells which are in complete continuity with one another. These epithelial cells are arranged in a papillomatous pattern. The surface of the epithelium shows various degrees of keratinization and parakeratosis and within the center of some of the columns and mass of cells epithelial pearl formation is seen. Everywhere the epithelial cells maintain a basement membrane and there is no evidence of invasion. The epithelial cells show the normal degree of variation in maturity of cells but no unusual pleomorphism, and mitotic figures are extremely infrequent.

Diagnosis: Benign nasal papilloma of the nasal septum. Pathology S. P. 46.2501.

The postoperative course of this patient has been uneventful and he has been seen regularly at one month, three month and, lately, six month intervals. He was last seen in May, 1949, two and one-half years after his original excision and there was no evidence of recurrence of the lesion. He is bothered during cold and dry weather by excessive crusting within his nose, but this is easily controlled by means of steam inhalations and the use of vaseline locally in his nose.

Case 6: J. F. S., male, age 72; Unit 169673. The patient presented himself at the University of California Hospital, Out-Patient Department, on Aug. 22, 1949, complaining of a small growth in the left nostril, which bleeds readily, of three months' duration. Examination revealed a small pedunculated, 4 mm. warty appearing lesion at the upper angle of the left nasal vestibule. The lesion was excised locally, under local anesthesia, and the base cauterized with electrocoagulation. The remainder of the ear, nose and throat examination was essentially normal.

Pathological examination reveals a small 3 mm. mass of tissue composed of piled-up and keratotic superficial epithelium and early pearl formation. There are strands of rather anaplastic appearing squamous cells throughout the tumor mass.

Diagnosis: Squamous cell carcinoma of the left nasal vestibule.

Examination in the outpatient department on Sept. 7, two weeks after excision, reveals the area to be entirely healed and free from induration or any evidence of recurrence. It is planned to observe this patient for evidence of recurrence.

Case 7: W. S., white female, age 45; Neo. 1012. This patient was first seen in the office on July 25, 1949, complaining of discharge of bloody mucosa from the left side of her nose of one week's duration.

Examination of the nose revealed a 1 x 1 cm. flat, pedunculated polyp which was attached to the midportion of the nasal septum to the left

ever, subsequent spinal stains revealed that the pale pink fibres gave the characteristic reactions for glial tissue, staining blue with phosphotungstic acid and yellow with Van Geisen's stain. They did not give the characteristic reaction of reticulum with silver. The final diagnosis was atrophy with glial tissue.

The patient was last seen on June 27, 1949, at the age of three years without evidence of recurrence.

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The patient was last seen on June 27, 1949, at the age of three years without evidence of recurrence.

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While it is desirable and necessary to demonstrate the existence and potential magnitude of a deleterious effect such as the hearing loss produced by gunfire noise, it is more important to evaluate the preventive measures which may minimize or eliminate the damage. For this reason it was decided that a field test of various types of ear protectors should be performed.

Earplugs were selected for evaluation in this project because they were apparently the most convenient form of ear protection. This was indicated by the fact that men on the firing line frequently used cotton plugs in their external ear canals and have been known to use many ingenious and subjectively more effective substitutes such as 45 calibre bullets or pencil erasers. The types of earplugs which were chosen for the project had been previously tested and[8] were readily available.

METHODS.

In order to determine the effect on the hearing from repeated exposure to gunfire, audiograms were taken on 187 instructors who had served on the firing ranges for from one to 14 months. These ranges included the skeet, moving base, hightower, ground, malfunction and air ranges, which will be discussed in more detail below. Audiograms were also taken on a control group of 184 enlisted students who had not yet begun active gunnery training.

Some of the qualifications of the individuals in the student and instructor groups which could easily distort the comparison of hearing acuity differences may be evaluated by the following. All the subjects in both groups had passed the same conversational voice hearing test for aerial gunners which *presumably* screened out men with gross hearing losses.

The average age of the students was 21.4 years and that of the instructors was 24.1 years. The mode of age for instructors was about two years greater than that for the students and fell within the 21-23 year age group.

A routine ear examination showed that the amount of scarring of the tympanic membranes was not marked among either the instructors or students and that there were no cases of chronic retraction of the drum. A few students were eliminated from the series because of the presence of aero-otitis media which had been incurred during their indoctrination in the altitude chamber. It was noted that the instructors had a high incidence of injection of the tympanic membranes especially when only a few hours' recovery period had been allowed. This was believed to be evidence of trauma inflicted by the concussion blast of gunfire.

While the students had been close to the noise of gunfire (50 and 30 calibre machine guns and 45 calibre pistols) at least in their basic training, there had been a minimum of three weeks since their last exposure. The instructors, on the other hand, had been exposed daily to either shotguns or 30 and 50 calibre machine gun noise for a prolonged period. It was possible to permit an auditory recovery period of 24 to 48 hours before the hearing curves were taken on 106 of this latter group (skeet, moving base, hightower, malfunction No. 2 and air range instructors), but the remainder (ground range and malfunction No. 1 instructors) had left the firing line three to six hours prior to the hearing test.

In order to evaluate the hearing recovery rate following exposure to gunfire, 11 instructors who had been firing 50 calibre machine guns each day every third week were repeatedly tested by audiometric methods during one recovery period of two weeks. Their selection was based upon their convenient schedule rather than on deficient hearing or other factors.

While all of the instructors had initially passed through the flexible gunnery school as students and had thus fired each type of gun on all the various types of ranges, their most prolonged exposure to repeated gunfire had been limited usually to the range to which each individual had been subsequently assigned as instructor. Since there were definite differences in the physical arrangement and sound intensity of each of the seven types of ranges, a brief description of each range will be given.

Skeet Range (Shotgun): These ranges were laid out in the standard fashion with high board fences intervening between each range and its neighbors. The fences tended to reflect the noise when a gun was fired from any position close to them. Almost all of the 12-gauge shotguns employed were fitted with compensators which apparently project a great deal of noise backward. The instructor stood to the side of and facing the student so that he was exposed to the concussion wave. Few of the instructors wore ear protection of any kind. This was also true on the other shotgun ranges.

Moving Base Range (Shotgun): On these ranges the student fired from the side of a truck which was moving at the rate of about 25 miles per hour. The instructor was stationed by the side of the student.

Hightower Range (Shotgun): These ranges were provided with Martin turrets which were fitted with twin shotguns and mounted on parked trucks. The instructor was located on a platform just behind and above the student, so that he received the maximum concussion only when the gun muzzles were markedly elevated.

Malfunction Range No. 1 (50 Calibre Machine Guns): Fixed machine guns were fired on this range with their muzzles protruding through small ports in a wooden wall. A wooden roof covered the guns and, while it extended beyond the plane of the front wall, there was an open space between the two structures which allowed the concussion waves to enter. The blast was painful to the ears unless some type of ear protection was worn. This was true on all the ground ranges where 30 and 50 caliber machine guns were being fired. Both the instructors and students recognized this fact and usually used earplugs of cotton.

Malfunction Range No. 2 (50 Calibre Machine Guns): This was similar to the range just described except for the fact that there was no wooden front wall. It was subjectively the noisiest range encountered and the concussion waves reflected from the roof could be felt by anyone beneath the shed as a powerful slap on top of the head.

Ground Range (30 and 50 Calibre Machine Guns): The machine guns used on this range were either hand held or mounted in Martin or Sperry ball turrets. The turrets were mounted on parked trucks and none of the guns was covered by a roof. The instructor, in most instances, was stationed behind the student. The concussion from a neighboring gun was frequently louder than that from the gun at which the instructor was located.

Air Range (30 and 50 Calibre Machine Guns): In aircraft, the students fired from the different types of turrets and from the hand held waist guns. The instructors (air specialists) varied their positions in accord with the constantly changing circumstances. Subjectively, there was a relatively small difference between the noise levels of the aircraft and that of the machine guns. The noise level of the gunfire heard in the plane was perhaps decreased by the presence of an intervening structure, *i.e.*, the skin of the fuselage, and by the carrying off of noise in the slipstream.

Each instructor and student filled out a questionnaire concerning exposure to noise, the use of earplugs and any aural difficulties. In addition to this, a routine examination of all tympanic membranes was performed and the results recorded.

In order to minimize the errors due to the subject's lack of familiarity with the hearing test procedure, each student and instructor was given two complete audiometric examinations, and only the second of these was recorded. The preliminary or indoctrination tests were performed by two enlisted men who had been recently trained for this work. The final audiograms were taken by two trained audiometrists who had had many months of experience in this type of work.

No sound conditioned rooms were available. In order to obtain a satisfactorily low ambient noise level during the testing, it was necessary to take the audiograms in a building which was some distance from any noise source. Testing during the night hours was the only possible means of avoiding the noise from planes in flight. The ambient noise in the testing rooms varied from 26 to 42 db as measured on a General Radio Sound Level Meter, Type 759-B ("A" weighting). The type of audiometer employed was the Maico D-5 and the two instruments used for the final test had been calibrated just prior to the initiation of the project.

In the study of earplugs as protectors, audiograms were taken on 331 gunnery students at the school of flexible gunnery before the beginning and during the final days of a six weeks' course. Every subject had his ears examined otoscopically and filled out a questionnaire before each of these hearing tests. Sixty-six of the men were asked to act as controls and wear no ear protection during the training period. Each of the remaining students was assigned a specific type of earplug and was requested to wear it whenever he was exposed to the noise of gunfire.

Five types or modifications of earplugs were issued. The students who were to use these plugs were shown how to insert them so that a maximum of hearing protection would be obtained. The plugs may be briefly described as follows:

1. *Sepco Ear Protector** consists of a bullet-shaped shell of rubber which is filled with small pieces of airfoam rubber and is sealed at its base by an airfoam rubber stopper. A flange around the shell prevents overinsertion into the ear canal.[8,9]

2. *NDRC V-51 Ear Warden* (Neoprene)† is mushroom-shaped and has a small tab protruding from its base, which facilitates removal from the ear canal.[8,10]

3. *Cotton Twists* are prepared individually from bulk cotton so that they fit snugly in the external canals and the concha of the ears.

4. *Lubricated Cotton Twists* differ from the above only in that the cotton is moistened with a water-soluble jelly.

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*Safety Ear Protector Co., Los Angeles, Calif.
†Psycho-Acoustic Laboratory, Harvard University, Cambridge, Mass.

5. *Lubricated Cotton Rolls* consist of short lengths of three-eighths inch dental rolls coated with water-soluble jelly.

The gunnery students who acted as subjects for this project were members of a single class, and all took the flexible gunnery course during the same six weeks' period. They had been exposed to the noise of gunfire during their basic training, but at least three weeks had elapsed between that exposure and the beginning of this study. The conversational voice hearing test which is required of all gunners had *presumably* eliminated men with gross hearing defects. The subjects ranged from 18 to 36 years of age and the average was 21.9 years.

The routine ear examination revealed that several subjects had aero-otitis media. These were eliminated from this series because of the temporary deafness involved. Cerumen was removed from the ears of the few men who had complete occlusion of the external canal. No other ear conditions were encountered which obviously would affect the validity of comparing the initial and final audiograms.

It has been pointed out above that of the 331 students, 66 were designated as controls. The remaining 265 subjects were divided into five groups according to the type of earplugs employed. Each of these groups will hereafter be designated by the name of the earplug which was used by all of the subjects in that specific group (*i.e.*, the Sepco group, the cotton twists group, etc.).

The selection of subjects to act as controls or to test a particular type of earplug was determined by their order of appearance for the initial hearing test. Quotas for each group had been estimated from the total number of men in the class and these quotas were filled for the control group, and then each successive group. Because of circumstances beyond our control, a comparatively small number were available for testing lubricated cotton rolls. The actual number of subjects in each group who completed the tests is as follows:

1. Control Group	66 subjects
2. Sepco Ear Protector Group	61 subjects
3. NDRC V-51R Ear Warden Group	58 subjects
4. Cotton Twist Group	56 subjects
5. Lubricated Cotton Twist Group	61 subjects
6. Lubricated Cotton Rolls Group	29 subjects

The audiograms in this part of the study were taken on the same audiometers, by the same operators and under the same conditions previously described.

It is impossible to evaluate the exact amount of exposure to gunfire which these subjects experienced during the course, for, although they fired in rotation on the same or similar ranges, they were exposed to a variable amount of noise from their own and other men's guns. Each student was supposed to fire a minimum of 600 shotgun shells, 650 50 calibre machine gun shells and 2,400 30 calibre machine gun shells. During the first two weeks most of the firing took place on the shotgun ranges. The next two weeks, 30 and 50 calibre machine guns were fired on the ground ranges, and during the final two weeks either 30 or 50 calibre machine guns were fired from aircraft.

RESULTS.

The comparison of the average audiograms of the 187 instructors with that of the 184 students (for both the right and left ears) is shown in Chart 1. A greater high tone loss of hearing among the instructors is indicated by the statistically significant differences which occur at the frequencies 2,048 through 11,584 cps. The losses are more apparent in the left ear and are most marked at and above the 4,096 cps. frequency.

In order to evaluate the detrimental effects of the various types of firing on hearing, seven average audiometric curves were compiled which represent the hearing of the instructors on each type of range. These were compared with the control (students) average audiogram. The data for the right and left

CHART I

COMPARISON OF AVERAGE AUDITORY THRESHOLDS OF GUNNERY INSTRUCTORS WITH GUNNERY STUDENTS

SUBJECTS	NUMBER	FREQUENCIES 128	256	512	1024	2048	2896	4096	5792	8192	11584
RIGHT EARS											
Instructors	187	10.0	6.8	8.5	6.9	6.3	25.0	35.3	35.7	30.8	39.4
Students	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		2.7	2.3	1.7	2.0	5.1	10.2	15.5	15.8	15.3	13.7
LEFT EARS											
Instructors	187	8.7	5.4	8.5	7.2	8.4	30.6	40.1	37.3	31.1	38.1
Students	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		0.8	1.1	1.7	2.6	6.2	14.0	20.0	19.1	19.6	17.3

NOTE: A statistically significant difference ($P < .01$) is indicated by a line under the difference.

ears were handled separately, and again it was noted that in every instance the hearing differences were slightly more marked in the left ears (see Charts 2 and 3).

It is apparent that the hearing of the air specialists deviates least of all from that of the control group. On the other

CHART 2

COMPARISON OF AVERAGE THRESHOLDS (IN DECIBELS) OF SEVEN GROUPS OF GUNNERY INSTRUCTORS WITH ONE GROUP OF STUDENTS (CONTROL)

RIGHT EARS

		FREQUENCIES									
Subjects	Number	128	256	512	1024	2048	2896	4096	5792	8192	11584
Skeet Instructors	12	13.3	9.6	13.8	12.9	12.5	48.3	56.7	50.4	45.0	52.1
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		6.0	5.1	7.0	8.0	11.3	33.5	36.9	30.5	29.5	26.4
Moving Base Instructors	10	15.5	11.0	11.0	9.0	8.5	30.0	44.0	45.5	41.0	50.5
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		8.2	6.5	4.2	4.1	7.3	15.2	24.2	25.6	25.5	24.8
High Tower Instructors	13	12.3	10.0	13.1	8.1	3.1	22.3	48.1	45.8	38.5	46.5
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		5.0	5.5	6.3	3.2	1.9	7.5	28.3	25.9	23.0	20.8
Malfunction #1 Instructors	14	9.6	7.9	9.6	6.8	2.5	21.4	33.2	37.9	32.9	44.6
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		2.3	3.4	2.8	1.9	1.3	6.6	13.4	18.0	17.4	18.9
Malfunction #2 Instructors	22	13.0	8.9	11.6	13.6	15.7	33.0	36.6	38.9	33.6	41.1
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		5.7	4.4	4.8	8.7	14.5	18.2	16.8	19.0	18.1	15.4
Ground Range Instructors	67	8.4	4.9	7.0	5.5	5.7	25.3	35.4	36.3	29.9	37.7
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		1.1	0.4	0.2	0.6	4.5	10.5	15.6	16.4	14.4	12.0
Air Specialists	49	8.5	5.9	5.8	3.7	2.7	15.9	24.9	24.6	22.7	32.3
Students (Control)	184	7.3	4.5	6.8	4.9	1.2	14.8	19.8	19.9	15.5	25.7
Difference		1.2	1.4	−1.0	−1.2	1.5	1.1	5.1	4.7	7.2	6.6

NOTE: A statistically significant difference ($P < .01$) is indicated by a line under the difference.

hand, the skeet range instructors have the most marked hearing loss. The other men who were exposed to shotgun fire (moving base and hightower) also show a loss of hearing for high tones which apparently exceeds the amount incurred by the instructors on the ground, malfunction and air ranges. It

CHART 3

COMPARISON OF AVERAGE THRESHOLDS (IN DECIBELS) OF SEVEN GROUPS OF GUNNERY INSTRUCTORS
WITH ONE GROUP OF GUNNERY STUDENTS (CONTROL)

LEFT EARS

		FREQUENCIES									
Subjects	Number	128	256	512	1024	2048	2896	4096	5792	8192	11584
SKEET Instructors	12	9.2	3.8	8.8	8.3	13.8	52.9	66.7	56.3	54.6	56.3
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		1.3	-0.5	2.0	3.7	11.6	36.3	46.6	38.1	43.1	35.5
MOVING BASE Instructors	10	16.0	11.0	14.5	15.5	15.5	51.0	60.0	55.5	52.0	66.5
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		8.1	6.7	7.7	10.9	13.3	34.4	39.9	37.3	40.5	45.7
HIGH TOWER Instructors	13	10.8	6.5	15.4	13.5	13.1	36.9	47.7	41.2	40.4	46.9
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		2.9	2.2	8.6	8.9	10.9	20.3	27.6	23.0	28.9	26.1
MALFUNCTION #1 Instructors	14	8.2	5.4	7.9	4.6	5.4	18.9	31.8	39.3	35.7	44.3
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		0.3	1.1	1.1	0.0	3.2	2.3	11.7	21.1	24.2	23.5
MALFUNCTION #2 Instructors	22	11.1	9.5	12.3	11.8	16.4	34.3	45.5	37.0	31.1	35.5
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		3.2	5.2	5.5	7.2	14.2	17.7	25.4	18.8	19.6	14.7
GROUND RANGE Instructors	67	8.1	4.6	7.5	5.6	6.2	28.7	37.8	38.8	28.1	34.5
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		0.2	0.3	0.7	1.0	4.0	12.1	17.7	20.6	16.6	13.7
AIR SPECIALISTS	19	6.2	3.5	5.4	4.3	4.6	23.5	30.7	25.4	21.4	29.8
Students (Control)	184	7.9	4.3	6.8	4.6	2.2	16.6	20.1	18.2	11.5	20.8
Difference		-1.7	-0.8	-1.4	-0.3	2.4	6.9	10.6	7.2	9.9	9.0

NOTE: A statistically significant difference ($P < .01$) is indicated by a line under the difference.

may be stated that the average hearing curves for the instructors on each of the ranges show decided losses of hearing for the higher tones and that, with the exception of the air specialists, there is a statistically significant difference between many of these losses and those of the control group.

CHART 4

COMPARISON OF AVERAGE AUDITORY THRESHOLDS OF GUNNERY INSTRUCTORS WITH GUNNERY STUDENTS BY AGE GROUP

Age Group	Subjects	Number	FREQUENCIES									
			128	256	512	1024	2048	2896	4096	5792	8192	11584
RIGHT EARS												
18-20 yrs.	Instructors	26	8.1	5.2	5.6	4.4	1.9	17.5	33.3	31.7	25.8	28.8
	Students	150	7.1	4.0	5.8	3.9	0.2	13.4	19.2	19.4	14.0	23.4
	Difference		1.0	1.2	-0.2	0.5	1.7	4.1	14.1	12.3	11.8	5.4
21-23 yrs.	Instructors	75	10.2	6.6	7.9	6.9	6.2	14.2	35.8	37.1	33.3	41.2
	Students	89	6.8	4.4	5.7	4.0	0.9	12.9	12.9	18.0	14.4	23.8
	Difference		3.4	2.2	2.2	2.9	5.3	1.3	22.9	19.1	18.9	17.4
24-26 yrs.	Instructors	38	7.8	5.0	6.3	4.9	5.8	8.6	32.2	34.6	30.4	39.2
	Students	62	6.7	4.0	6.6	3.9	0.8	16.9	23.3	21.7	17.3	28.1
	Difference		1.1	1.0	-0.3	1.0	5.0	-8.3	8.9	12.9	13.1	11.1
27-29 yrs.	Instructors	29	14.0	11.0	15.0	11.9	8.6	10.2	34.8	31.7	26.7	41.0
	Students	19	9.7	5.8	7.1	6.8	1.8	12.6	15.5	22.1	16.6	22.6
	Difference		4.3	5.2	7.9	5.1	6.8	-2.4	19.3	9.6	10.1	18.4
LEFT EARS												
18-20 yrs.	Instructors	26	7.1	3.3	6.9	4.8	1.9	16.5	31.9	33.9	24.2	31.0
	Students	150	7.5	4.2	6.1	4.1	1.1	14.2	17.4	16.6	10.1	16.6
	Difference		-0.4	-0.9	0.8	0.7	0.8	2.3	14.5	17.3	14.1	14.4
21-23 yrs.	Instructors	75	9.3	6.4	9.1	8.1	9.8	34.1	42.7	38.7	33.5	39.5
	Students	89	7.6	4.3	6.3	4.2	2.4	16.1	21.7	19.3	12.1	19.7
	Difference		1.7	2.1	2.8	3.9	7.4	18.0	21.0	19.4	21.4	19.8
24-26 yrs.	Instructors	38	6.7	3.3	5.9	4.2	5.9	29.3	38.8	38.0	30.1	38.2
	Students	62	6.7	2.5	5.2	3.0	1.3	17.3	23.0	18.9	13.7	23.6
	Difference		0.0	0.8	0.7	1.2	4.6	12.0	15.8	19.1	16.4	14.6
27-29 yrs.	Instructors	29	10.3	6.7	11.7	10.5	8.6	28.5	37.8	31.9	30.5	38.4
	Students	19	8.9	4.7	6.6	4.2	2.1	14.7	19.7	18.4	12.4	21.8
	Difference		1.4	2.0	5.1	6.3	6.5	13.8	18.1	13.5	18.1	16.6

NOTE: A statistically significant difference ($P < .01$) is indicated by a line under the difference.

The comparison of average auditory thresholds of instructors with students by age groups is shown on Chart 4.* It is apparent that there was a predominance of students in the youngest group (18-20 years) and of instructors in the oldest group (27-29 years). The least number of significant differences between the average audiograms of the students and instructors occur in the oldest and youngest age groups, while the greatest number occur in the 21-23 years of age group. There is no regular progression of hearing losses for the high tones in the progressively older groups of either students or instructors.

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*Since it was recognized that the older average age of the instructors might play an important rôle in the production of greater average hearing losses and the number of students in the older age group was not sufficient to provide a valid comparison, the audiograms of 136 additional students were used in making an analysis of the age factor. These students were comparable with the original group and audiograms were taken under the same conditions.

The 11 subjects who were followed with repeated audiograms during a recovery period of two weeks were initially tested one-half to two hours after they had ceased firing. Subsequent audiograms were taken at the end of 24 hours, 48 hours and two weeks. The average audiograms for these recovery periods are shown in Fig. 1. Despite the fact that these hearing tests do not show a uniformly progressive recovery, it would appear that the major portion of temporary high tone hearing loss had disappeared within 24 to 48 hours after the last exposure to gunfire noise.

HEARING RECOVERY—AVERAGE AUDIOGRAMS OF LEFT EARS (11 Subjects).

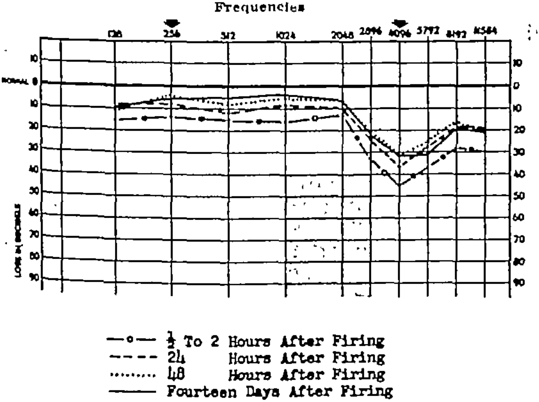

Fig. 1.

CHART 5
AVERAGE AUDIOMETRIC HEARING CHANGES DURING GUNNERY TRAINING IN DECIBELS
LEFT EARS

			FREQUENCIES									
GROUPS	NO. EARS		128	256	512	1024	2048	2896	4096	5792	8192	11584
Control	66	After Training	8.0	4.6	6.1	4.7	4.3	20.7	28.0	25.2	18.6	25.8
		Before Training	8.3	4.7	6.5	3.8	2.1	15.2	18.9	16.4	11.8	20.5
		Difference	-0.3	-0.1	-0.4	0.9	2.2	5.5	9.1	8.8	6.8	5.3
Sepco Ear Protector	61	After Training	6.5	3.1	4.5	3.0	1.6	15.0	20.2	19.5	13.6	19.7
		Before Training	7.1	3.5	6.2	3.9	1.3	14.5	18.5	17.5	10.3	19.7
		Difference	-0.6	-0.4	-1.6	-0.9	0.3	0.5	1.7	2.0	3.3	0.0
NDRC V-51R Ear Warden	58	After Training	6.6	3.2	5.6	4.1	1.3	14.0	19.8	16.8	12.9	19.1
		Before Training	6.4	3.0	5.9	4.0	1.5	15.8	19.3	16.8	10.7	21.0
		Difference	0.2	0.2	-0.3	0.1	-0.2	-1.8	0.5	0.0	2.2	-1.9
Cotton Twist	56	After Training	7.5	4.8	6.2	5.2	4.9	19.7	24.1	23.8	17.5	20.7
		Before Training	8.6	4.6	7.1	5.2	3.5	18.4	22.6	20.3	11.7	17.9
		Difference	-1.1	0.2	-0.9	0.0	1.4	1.3	1.5	3.5	5.8	2.8
Lubricated Cotton Twist	61	After Training	7.3	4.3	5.7	4.0	0.9	14.3	23.1	19.5	14.8	17.2
		Before Training	6.9	3.4	4.8	3.5	0.7	14.2	21.4	19.1	11.9	16.0
		Difference	0.4	0.9	0.9	0.5	0.2	0.1	1.7	0.4	2.9	1.2
Lubricated Cotton Rolls	29	After Training	7.2	3.5	6.4	4.3	0.5	16.6	26.7	22.1	18.8	25.2
		Before Training	7.8	4.8	6.4	3.8	0.3	16.7	21.9	19.8	15.2	22.2
		Difference	-0.6	-1.3	0.0	0.5	0.2	-0.1	4.8	2.3	3.6	3.0

NOTE: A Statistically significant difference ($P < .01$) is indicated by a line under the difference.

In the earplug study a comparison of the average audiograms of the control group before and after the course (see Charts 5 and 6) demonstrates that statistically significant hearing losses occurred during the exposure to the noise of gunfire and were produced in the higher frequencies (2,896 to

CHART 6

AVERAGE AUDIOMETRIC HEARING CHANGES DURING GUNNERY TRAINING IN DECIBELS

RIGHT EARS

GROUPS	NO. EARS		FREQUENCIES 128	256	512	1024	2048	2896	4096	5792	8192	11584
Control	66	After Training	6.9	4.1	5.8	4.0	0.8	16.7	24.9	25.3	21.4	29.9
		Before Training	7.8	4.4	6.1	3.7	-0.6	13.6	19.5	20.8	15.5	24.6
		Difference	-0.9	-0.3	-0.3	0.3	1.4	3.1	5.4	4.5	5.9	5.3
Sepco Ear Protector	61	After Training	10.2	5.1	6.6	4.2	2.1	16.4	19.7	22.6	17.5	29.6
		Before Training	6.4	3.9	6.2	2.5	-0.7	17.3	19.8	20.5	15.7	28.8
		Difference	3.8	1.2	0.4	1.6	2.8	-0.9	-0.1	2.1	1.8	0.8
NDRC V-51R Ear Warden	58	After Training	6.6	3.4	5.9	5.9	1.9	16.0	22.5	21.6	16.7	24.7
		Before Training	6.3	4.1	6.6	5.1	2.2	15.3	21.1	20.3	18.5	27.1
		Difference	0.3	-0.7	-0.7	0.8	-0.3	0.7	1.4	1.3	-1.8	-2.4
Cotton Twist	56	After Training	7.9	4.6	5.8	4.8	2.9	18.8	22.8	22.9	17.8	26.2
		Before Training	7.3	4.6	6.8	5.9	2.2	16.4	20.1	20.0	13.8	25.9
		Difference	0.6	0.0	-1.0	-1.1	0.7	2.4	2.7	2.9	4.0	0.3
Lubricated Cotton Twist	61	After Training	11.6	6.5	7.5	6.5	2.3	17.2	24.3	22.5	18.7	26.6
		Before Training	6.9	3.6	3.9	3.7	1.2	14.8	23.9	23.8	19.5	27.2
		Difference	4.7	2.9	3.6	2.8	1.1	2.4	0.4	-1.3	-0.8	-0.6
Lubricated Cotton Rolls	29	After Training	7.8	4.0	4.5	2.9	0.9	17.2	22.2	18.3	14.8	21.9
		Before Training	8.1	5.5	6.9	4.8	0.5	15.0	22.2	19.3	11.6	22.2
		Difference	-0.3	-1.5	-2.4	-1.9	0.4	2.2	0.0	-1.0	3.2	-0.3

NOTE: A statistically significant difference (P<.01) is indicated by a line under the difference.

11,584 cps.). In amount these losses range from about 3 to 9 db and are somewhat more marked in the left ears.

Similar comparisons of the average audiograms of the five groups who tested the different types of earplugs reveal only one statistically significant loss (see Charts 5 and 6). This appears at the 8,192 cps. frequency in the left ears of the subjects who wore Sepco earplugs, and is considered to be a possible chance variation.

DISCUSSION.

Since no audiograms had been taken on the instructors prior to their entrance into the gunnery course, it was necessary to use a control group composed of students who were about to enter gunnery training. It is believed that the mean hearing of these students may be considered grossly comparable to the average hearing of these instructors prior to their entrance into training, in that the members of both groups had been exposed to gunfire noise of basic training and had subsequently passed the same conversational voice hearing tests.

It is difficult to draw conclusions concerning the influence that age may have played in the very apparent differences between the mean audiograms of students and instructors (see Chart 1). For example, there is no adequate explanation for the small number of significant differences in hearing in the youngest and oldest groups and the preponderance of these differences in the 21-23 years of age group (see Chart 4). It would appear from previous studies[5,6] that the small mean age difference (two and one-half years) that exists between the students and instructors would hardly account for a great amount of the discrepancy between these average audiograms. This assumption is supported by the fact that there is no regular increase of hearing loss in the progressively older groups of either students or instructors (see Chart 4).

As pointed out previously, the student hearing tests were taken after a recovery period of at least three weeks, but the instructors were tested three to 48 hours after they had last fired. The temporary hearing loss which must have been present in this latter group composes at least a part of the comparative differences between the average audiogram of the students and that of the instructors (see Chart 1). The exact amount of this temporary hearing loss cannot be determined, but there are certain factors which may aid in a gross evaluation. The audiograms of 106 instructors who were tested after a recovery period of from 24 to 48 hours should include only a small amount of temporary hearing loss, for it was shown on a small control series (see Fig. 1) that the major portion of high tone hearing recovery took place within a period of 24 to 48 hours. The remaining 81 instructors were tested from three to six hours after firing and undoubtedly their audiograms show a greater amount of temporary loss.

It has been pointed out that the air specialists demonstrated less average hearing loss than any of the groups of instructors from the other ranges (see Charts 2 and 3). This may indicate that the high intensity of ambient aircraft noise causes a muscular splinting of the sound conducting mechanism in the middle ear which partially protects the cochlea against the relatively greater intensity of gunfire noise.

Despite the fact that the instructors on the skeet, moving base and hightower ranges had a 24 to 48 hour recovery period before being tested, their average audiograms show more marked hearing losses than any of the other instructor groups (see Charts 2 and 3). The explanation for this would seem to be that the noise of shotgun firing was not sufficiently intense to cause marked aural discomfort and, therefore, did not force the instructors to wear ear protection.

The fact that the group of subjects who wore ear protection in the study of earplugs incurred insignificant average hearing losses while the control group incurred significant losses would infer that any one of these five types of earplugs is capable of affording adequate protection against the amount of noise encountered in such a flexible gunnery course; however, it is possible that the small hearing losses might have become significantly larger in any or all of these groups if the duration of exposure to, or the intensity of, the gunfire noise had been markedly increased.

Since a longer or shorter final recovery period for the majority of subjects in any one group might have diminished

CHART 7

NUMBER OF SUBJECTS IN EACH GROUP TESTED ONE, TWO, OR THREE DAYS AFTER FIRING

RECOVERY PERIOD	NUMBER OF SUBJECTS					
	CONTROL GROUP	SEPCO GROUP	NDRC V-51R EAR WARDEN GROUP	COTTON TWIST GROUP	LUBRICATED COTTON TWIST GROUP	LUBRICATED COTTON ROLLS GROUP
ONE DAY	34	29	43	39	44	11
TWO DAYS	27	29	15	12	13	18
THREE DAYS	5	3	0	5	4	0

or increased the average hearing loss for that group and since a single uniform recovery period was not permitted all the subjects before their final audiometric examination, the number of students in each group who were tested one, two or three days after firing must be considered. Inspection of Chart 7 reveals that the number of men in each group who were tested after any one of these three recovery periods are not proportionately comparable for all groups. The following however, should be pointed out. The control group was the only one which showed significant hearing losses (see Charts 5 and 6) and approximately half of the men comprising this group were tested two or three days after firing. On the other hand, the NDRC V-51R Ear Warden, cotton twist, lubricated cotton twist and Sepco groups did not show significant hearing losses, although well over 50 per cent of the subjects in each of the first three groups and almost half of the subjects in the Sepco group were permitted a recovery period of only one day. There is no doubt that the longer period (two days) for approximately 60 per cent of the students in the lubricated cotton rolls group may have reduced the average hearing losses in this group.

The questionnaires revealed that almost all of the controls were forced by discomfort to wear cotton in their ears on one exceedingly noisy range where 50 calibre machine guns were fired from beneath sheds. The use of this protection probably reduced the magnitude of the average hearing losses in this group but the exact amount cannot be estimated.

It was hoped that the results of this project would demonstrate the relative protection afforded by each of the five types of earplugs but, obviously, the hearing losses were not of sufficient magnitude to permit significant differentiation.

CONCLUSIONS.

a. The noise of gunfire (50 calibre machine gun and shotgun) produced a definite loss of hearing for high tones (2,048-11,584 cps.) among gunnery instructors (see Chart 1).

b. A major portion of the temporary high tone hearing loss which is present immediately after exposure to the noise of gunfire is recovered in the subsequent 24 to 48 hours (see Fig. 1).

c. Significant high tone (2,896-11,584 cps.) hearing losses (3 to 9 db) were produced in the average audiograms of a of neisseria have been reported as isolated from animals. Their method of obtaining the cultures is described and a study of the various characteristics tabulated.

DISEASES.

Rubin[7] points out that the nasopharynx is a definite anatomic region containing lymphoid tissue which is known as the adenoid. The latter may become acutely infected just as the tonsils or the pharyngeal lymphoid tissue. Such infection produces varying degrees of malaise and sore throat, symptoms for which a cause may be difficult to find unless the nasopharynx is examined. This condition represents true acute pharyngitis. Rubin thinks that the latter term is too often used indiscriminately to refer to unidentified inflammation of the upper respiratory tract and should be reserved for specific infections of the nasopharynx itself.

Zak and Cisneros[8] describe what they believe to be the first case of psoriasis of the hypopharynx to be reported in the literature. The lesion was discovered at necropsy following death from massive pulmonary emobilization in a man 60 years old who died three weeks after craniotomy for removal of a supracellar meningioma. At necropsy, psoriatic lesions were noted on the left forearm and the wall of the hypopharynx. The section from the hypopharyngeal lesion revealed a large degree of accumulation and desquamation of nucleated squamous epithelial cells analogous to the parakeratosis of cutaneous psoriasis accompanied by a focal disturbance of the stratified epithelium. There was also a conspicuous accumulation of amorphous eosinophilic material in the submucosa. The absence of the usual psoriatic acanthosis is explained by the authors by the fact that the normally scant papillae of this region were flattened out and obliterated by this edematous infiltration. The edematous infiltration contained collagen and elastin bundles spread widely apart.

The Bremonds[9] report the case of a woman, 42 years old, who complained of pain, of five years' duration, in the left side of the pharynx in the tonsillar region, radiating toward the ear, maxilla and submaxillary region on the same side. The left tonsil was slightly enlarged; palpation of the anterior wall of the tonsillar bed was painful and revealed a hard body. The pain was completely relieved by removal of a portion of the stylohyoid ligament, which had undergone ossification.

Phelps and associates[10] report the results of a detailed study, from the standpoint of clinical and laboratory observations and response to three kinds of treatment, of 272 consecutive cases of streptococcal pharyngitis encountered at a large Air Force station hospital during a 19-day period. Throat cultures positive for streptococcal hemolyticus were obtained in 89.3 per cent of the series. Complications occurred in 14.3 per cent of the series. Penicillin was found to be more effective than sulfadiazine or symptomatic treatment, although sulfadiazine, like penicillin, shortened the period of hospitalization, hastened subsidence of edema and redness, and reduced the number of complications.

Swanston[11] presents a case of ocular pemphigus in a woman 80 years old. Incident and concomitant findings were bilateral epistaxis from Kiesselbach's plexus and nonspecific ulcers of the tonsillar pillars, epiglottis and arytenoids.

Kelley[12] presents a discussion of 10 consecutive cases of oral and pharyngeal inflammation in adults with Monilia albicans isolated as the only or the predominant organism. These patients were successfully treated by local applications of gentian violet and large doses of vitamin B complex orally for periods of one to 10 months with an average of 3.5 months. Contrary to the reports in the literature, the most conspicuous feature of these cases was the frequent association of "white furry" patches with "black furry" patches. Kelley believes that because of the common causative agent and the uniformly successful treatment there may be justification for considering these furry patches of all hues between white and black as belonging to the same clinical entity.

In an excellent article well worth reading in its original form, Davidson[13] calls attention to the fact that whereas numerous cases of abscesses of the retropharyngeal spaces have been reported in children, only 114 cases in adults have been reported in the literature. He reports eight additional cases from the records of Barnes Hospital — an incidence of one in 11,659 hospital admissions. He states that there are four potential spaces behind the pharynx; two peripharyngeal spaces extending from the carotid sheath on each side to the cricoid below and the upper border of the superior constriction above. They are divided in the midline by a fibrous septum. The postvisceral space lies between the prevertebral muscles and the peripharyngeal space and extends below into the posterior mediastinum. The prevertebral space exists between the vertebral and the prevertebral fascia. There are lymph nodes in the peripharyngeal space in children which are not usually present in adults. This is believed by some to account for the uncommon occurrence of abscesses of these spaces in adults.

Abscess of the prevertebral space is most commonly associated with tuberculosis of the cervical spine.

The commonest causes of abscesses of the retropharyngeal spaces are otitis and mastoiditis. Many possible methods by which otogenic pharyngeal abscesses are produced are discussed. Contrary to common belief, abscess of the retropharyngeal spaces is rarely due to foreign bodies. These abscesses are diagnosed by the history of dysphagia, pain in the throat, and dyspnea, and by the observation of a mass protruding from the posterior pharyngeal wall. The importance of a lateral Roentgenogram of the cervical spine must not be overlooked. The commonest complications of these abscesses are mediastinitis and hemorrhage from one of the larger vessels of the neck. The treatment consists in incision and drainage. There is a difference of opinion whether they should be drained externally or through the pharynx. Many reserve the external approach for those cases which are too low or too lateral for internal drainage. Two cases are reported.

Woodruff[14] discusses epistaxis and gives a brief analysis of 28 cases. He differentiates between bleeding from Kiesselbach's plexus (epistaxis digitorum) and bleeding from other areas of the nose which are associated with hypertensive cardiovascular disease; these he calls cardiovascular epistaxis. He has noted that in many of these patients the bleeding point is located posteriorly in the inferior meatus. He has observed dilated blood vessels in the posterior portion of the inferior meatus and the lateral nasopharyngeal wall which he calls the nasopharyngeal plexus. He advocates electrocoagulation of the bleeding point if accessible and tamponage with cotton tampons soaked in a mixture of tannic acid and antipyrine solutions when the bleeding point is not accessible. It was necessary to ligate the external carotid in four of his patients in order to control the hemorrhage; in three of these a septal deflection prevented adequate packing.

DIVERTICULA.

McNealy and Glassman[15] advocate two-stage pharyngoesophageal diverticulectomy for large diverticula which distort the structure from which it originates, and also for diverticula complicated by inflammation, ulceration and perforation. The first stage consists of freeing the sac, bringing it out laterally and performing a diverticulopexy. After two weeks or more, the second stage, resection of the sac, is performed. The technique of resection is described and illustrated and details of preoperative and postoperative care are given. McNealy and Glassman have never found it necessary to dilate the esophagus following diverticulectomy. They attribute this primarily to the transverse character of the resection and to the use of interrupted sutures of nontraumatized or crushed structures.

Blain[16] discusses the pathogenesis, complications, incidence and technique of surgical excision of pharyngoesophageal diverticula. He points out that these diverticula may now be safely removed in one stage unless the sac is extremely large. In the latter cases it is safer to correct the condition by a two-stage procedure because of the danger of infection in the widely open fascial planes of the neck and mediastinum. The two-stage operation allows time for walling off of these spaces by fibrinous exudate. Several cases are reported to illustrate the varied types of pharyngoesophageal diverticula and the necessity of selecting the procedure best suited to the specific case.

Rivas Diaz[17] reports a case of successful removal of a large pharyngoesophageal diverticulum in a female. The diverticulum caused the patient to have extreme difficulty on swallowing. The interesting feature of this case is the large size of the sac.

Waldapfel[18] reports two cases of patients with pharyngoesophageal diverticula whose symptoms were eliminated by dislocation of the sac. In the first case the sac was dislocated at the first stage and at a second stage it was resected. The second patient was treated by the one-stage procedure. Since symptoms were relieved in both cases, Waldapfel is of the opinion that in small and medium sized sacs, as well as some large ones, dislocation of the sac is sufficient. It is a safe procedure, easy to perform, causes minimal discomfort and short convalescence, and provides immediate postoperative normal feedings. If it is found that the sac needs to be resected, this can always be done at a later date. Waldapfel calls attention to the fact that resecting the sac does not always insure against recurrence. In his first case, despite resection, a new sac formed four years later.

Mino and associates[10] report a case of pharyngoesophageal diverticula of unusual size in a Negro 62 years old. They found no previous record of a larger diverticula of this type, and they know of no reported instance of a case with coexisting compression of the lungs. The patient sought medical attention because of weakness and dysphagia of about six months' duration. Roentgenograms revealed an enlarged oval mass lying in the mediastinum and upper part of the chest on the right. Its superior and inferior poles were opposite the first and sixth thoracic vertebrae, respectively. The lateral border of the mass was about 9 cm. from the midline of the chest, encroaching upon the pleural cavity and compressing the lung on the right side. Anteroposteriorly the mass was almost centrally located. Preliminary jejunostomy resulted in a gain of 26 pounds within three weeks. The diverticulum was removed in two stages because of its size. In the first stage the diverticulum was exposed through an incision beginning at the anterior edge of the right sternocleidomastoid muscle and extending from the sternum to the level of the hyoid bone. It was difficult to mobilize the diverticulum because of excessive adhesions. Dissection was facilitated by intermittent air injections through the lavaging tube. The diverticulum was implanted in the wound in a position which assured an obtuse angle at the junction of its neck with the esophagus. The second stage was performed 10 days after the first, at which time the diverticulum was excised and the esophageal orifice was closed. Roentgenograms nine months postoperatively revealed no abnormality of the pharynx, hypolarynx and esophagus.

In contradistinction to the current theory of a congenital weakness of the posterior pharyngeal wall, which fails to explain why pharyngoesophageal diverticula do not occur in children and are commoner in men than women, Horowitz[10] suggests that an acquired defect may account for the herniation. In support of this view is the fact that its occurrence in young persons has never been reported except for cases with organic stenosis of the upper end of the esophagus. Evidence is presented in support of King's concept that the presence of the lower end of the cricoid cartilage against the spine has a causative relationship in the development of these diverticula. This would explain why these diverticula occur more often in men, since their larynges are larger than women's and would be more firmly in contact with the spine, because men are more muscular than women. A fatal case of a perforated diverticulum in a woman 72 years old is described.

Shallow,[11] who advocates combined one-stage pharyngeal diverticulectomy, presents an analysis of 204 consecutive personal cases in which this method was employed. The salient features of the method consist in: *1.* cleansing and emptying the sac through the esophagoscope; and *2.* keeping the esophagoscope in the esophagus while the neck of the sac is being ligated, its stump transplanted and the pharynx repaired; this will maintain the normal anatomic position of the pharynx and upper part of the esophagus, a factor which favors normal function after healing has occurred. Esophagoscopic assistance permits cleansing of the sac from within, making the sac easy to identify, and prevents the development of stenosis and angulation at the site of repair. Attention is called to the necessity of meticulous repair of the pharyngeal wall. Shallow advocates anchoring the posterior portion of the suture line to the prevertebral fascia "because it strengthens the repair, obliterates the retropharyngeal space at this point, and erects a barrier against descending infection into the mediastinum." Shallow believes that the safety and efficacy of this method has been proved by the fact that in his series there were no cases of mediastinitis, there was only one recurrence and the mortality rate was 2.5 per cent in an age group averaging 60 years.

IRRADIATION.

Pool and associates[22] report their results in 150 patients with nasopharyngeal lymphoid hyperplasia and 20 patients with asthma treated during the war years with Roentgen-ray. Follow-up data were received from 72 per cent of the 170 cases; 34 per cent of these obtained some relief, 43 per cent were greatly improved, and 33 per cent obtained no relief. Several patients with asthma obtained remarkable relief following treatment with Roentgen-ray but they were receiving allergic treatment at the same time. There were no serious ill effects from the irradiation treatment. Of 20 children who received irradiation therapy for lymphoid hypertrophy, middle ear infection and deafness during the same period, 27 per cent greatly improved, 33 per cent received some benefit and 40 per cent were unimproved. The conclusion is drawn that "before the real value of irradiation as a therapeutic agent in allergic disorders can be determined, many more cases must be studied."

In a most excellent article on the treatment of infections of the upper respiratory tract, Proctor, Polvogt and Crowe[23] divide their discussion into three parts. They first discuss the physiologic functions of the upper respiratory tract by describing in minute detail the nasal mucosa, ciliary functions and lymphoid significance. The pathology of the upper respiratory tract is then discussed in considerable detail and irradiation of adenoids is described.

Results obtained by them have been highly beneficial. It must be clearly understood that irradiation of the nasopharynx with a radium applicator is no panacea, but in selected cases it is an invaluable therapeutic measure. Their technique of application as well as clinical indications is described. A report of the results obtained in competent hands emphasizes the tremendous value of adequate therapy properly applied. An important phase of this work is the chapter on the prevention of deafness in children.

This is by far the best article published recently on this subject and should be read by all interested in lymphoid hyperplasia of the nasopharynx.

In a study of the literature in the treatment of lymphoid tissue in the nasopharynx, Rius[24] found that 25,000 radium treatments were administered during World War II in an effort to control recurring aerotitis and infections of the nasopharynx with 90 per cent successful results. Rius discusses the technique and indications for the use of the radium applicator and reports a number of cases successfully treated in children and adults. This article, although serving an excellent purpose, adds nothing original to a subject already exhaustively discussed in the literature.

Scal[25] points out that irradiation therapy is not a treatment for deafness but for the elimination of conditions which may produce deafness. He has employed irradiation therapy with gratifying results in over 50 patients, most of whom had impaired hearing, repeated attacks of nasal colds and infected ears. He has also used this method in a group of children with allergic bronchitis and asthma, but the results in this group thus far have not been too encouraging. The method of application of radiation therapy is described and several typical cases are reported.

Collins and co-workers[26] observed considerable symptomatic improvement in 41 children followed from 18 months to two years after cessation of treatment by radon applications to the nasopharynx. They believe that to avoid overtreatment results should be judged symptomatically rather than by regression of lymphoid tissue. They consider the results of radon therapy to be superior to any obtained by use of antral lavage, nasal drops, vitamin therapy or any other form of treatment. No harmful effects were noted.

They advocate radon application for chronic conduction deafness of Eustachian origin and chronic nasal catarrh. Before irradiation is considered, they point out that the tonsils and adenoids should be removed when deemed necessary. They list among the chief dangers of irradiation: *1.* interference with centers of ossification, *2.* irradiation burns leading to scarring and occlusion of the Eustachian tubes, *3.* overdosage which may leave a wide and patulous Eustachian tube which is amenable to infections of the middle ear, and *4.* reduction of activity of the ciliated epithelium in the nasopharynx. They believe that these dangers can largely be avoided by use of small doses at widely spaced intervals. On the basis of the radiologic improvement in their series, they suggest that the mucosal thickening of the maxillary sinuses might be a secondary reaction to infected remnants in the nasopharynx, which are harboring pathogenic organisms and are also interfering with proper ventilation.

The use of radium, which is in general use in controlling nasopharyngeal lymphoid tissue, is discussed by Tyler,[27] who advocates its use in Eustachian tube obstruction due to lymphoid hyperplasia and in selected cases of postnasal discharge, recurrent rhinitis and sinusitis in children and in some cases of asthmatic bronchitis. No new information is contained in this article.

Robbins and Schulz[28] warn against the possible resultant dangers of the present vogue for treating hypertrophied lymphoid tissue in the nasopharynx with radium. They cite several cases of late radiation necrosis (sometimes not appearing for 10 to 20 years) which followed treatment of benign lesions with the same dosage as that recommended for use in the nasopharynx for hypertrophy of lymphoid tissue. In view of these dangers, they suggest as precautionary measures that the monel applicator not be employed routinely; that radiation be used cautiously in children and adults with benign lesions; that except in selected cases no more than erythema doses (determined by a given applicator) be given; and that the erythema dose can be determined, if necessary, by application to the examiner's skin.

Champion[29] reminds us that the nasopharynx is only part of the respiratory tract and is susceptible to infections occurring in other portions of the respiratory tract. It is abundantly supplied with lymphoid tissue which is not only susceptible to acute infections but also harbors chronic infections.

In spite of conscientious efforts at accurate removal, islands of lymphoid tissue are left *in situ*. These in time frequently become infected and hyperplasia occurs. This hyperplasia of the lymphoid tissue in time serves to obstruct the Eustachian orifice and interfere with good hearing. Surgical removal of this tissue is not always successful; for this reason radium applications to the nasopharynx are used. Champion describes the technique and indications which follow the usual pattern and claims that the beneficial effects of irradiation are due to the effect of radium on the germinal centers of the lymph nodes in which new cells are formed, the reduction of blockage in the lymphatic channels, and the resistance of irradiated cells to recurrent infections. He concludes that excellent results in the improvement of hearing have been obtained in some cases, and that occasionally irradiation will diminish the goblet and mucus glands of the mucosa of the nasopharynx, thereby diminishing the postnasal discharge.

Haggart[30] advises conservatism in the use of radium for irradiation of the nasopharynx. He points out that the gratifying results obtained in the treatment of deafness, chronic otitis media and infection with radium might be a temptation to employ larger doses at more frequent intervals, but this must be avoided. He believes that the use of radium should be restricted to properly selected cases and only safe dosages should be employed. He considers a safe routine the use of a 50 mg. applicator, inserted through the nose for 12 minutes to each side, not more than three or four times at two-week intervals within the year. Haggart mentions several potential dangers of nasopharyngeal irradiation which must be borne in mind when employing this procedure. The dosage employed must be right for the type of applicator used and the patient must be questioned before treatment is given to determine whether he has had radium or Roentgen-ray irradiation before.

Fisher[31] emphasizes the necessity of employing preventive measures in children if the number of deaf and partially deaf people in the United States is to be reduced. Such children show loss of hearing for high tones. For this reason it is necessary to test for high tones as well as lower tones if impaired hearing due to partial and intermittent obstruction of the Eustachian tube is to be detected early enough to institute preventive measures. In well selected cases irradiation therapy will produce diminution in size and a "smoothing out" of the nasopharyngeal lymphoid tissue which partially or completely obstructs the Eustachian tube orifices. During a two-year period 821 patients were examined in the "Better Hearing Clinic" at the Medical College of Alabama, 224 of whom showed loss of hearing due to Eustachian tube obstruction. Of 181 in whom irradiation therapy had been completed, hearing returned to normal in 93.

Thrift[32] discusses the introduction and development of radium therapy for lymphoid hyperplasia of the nasopharynx and gives a detailed description of the instrument used and the method of application. He has treated 65 cases, in 32 of whom treatment has been completed. Objectively, 80 to 85 per cent were improved and subjectively, 70 to 75 per cent. He noted a slow response in children, which he believes was due to selection of cases with too much tissue in the nasopharynx. He believes that in the presence of a large amount of tissue adenoidectomy should precede the use of the radium applicator. All the children in his series showed improvement. There were no complications in his cases.

Cutler and associates[33] present an analysis of 50 patients, including both adults and children, with nonmalignant lesions of the nasopharynx treated by irradiation according to the method of Crowe and followed for one year. From their study they observed that irradiation combined with other forms of therapy was useful in children with transitory impaired hearing associated with allergic symptoms. In these patients, associated paranasal infections are invariably present. As was expected the results of treatment were disappointing in cases of fixed impaired hearing of obvious otologic cause. Cutler and co-workers believe that radiation may be valuable in mild dysfunctions of the Eustachian tube, but point out that more experience will be necessary before this can be established.

Jones[34] calls attention to the beneficial effects of irradiation of the nasopharynx in properly selected cases by citing the results obtained by several authors as well as by himself in cases of impairment of hearing, aerotitis, tinnitus, fullness or pressure in the middle ear, focal infections, asthma and chronic purulent otitis media. He emphasizes the importance of treatment before irreversible changes have taken place and warns against the promiscuous use of irradiation with its unpleasant effects.

This is the sixth paper by Proctor[35] in a series of analyses of the various phases of the problem involved in a public health approach to the prevention of deafness. The material includes all children admitted to a clinic established in Hagerstown, Md., six years ago for the prevention of deafness in children. During the first four years the average follow-up for 687 patients was 16.4 months; 362 other patients were seen only once; 181 had been followed more than two years and 59 more than three years.

Analysis of this material revealed the following facts: *1.* in four of every five children with impaired hearing the difficulty was traceable to respiratory or aural infection, *2.* in many patients even if impairment had occurred it was reversible, *3* in 1,542 ears of 774 patients normal hearing was noted in 23 per cent of the ears when first seen and 49 per cent when last seen, *4.* over a five-year period there was a definite decrease in the number of patients with moderate to severe hearing impairment and chronic aural or sinus infection with each succeeding year.

Proctor believes that irradiation therapy should be confined to those with hearing impairment due to lymphoid tissue obstructing the tubal orifices, those with recurring acute respiratory or aural infections originating in infected adenoid tissue, and some patients with bronchial asthma on a basis of bacterial allergy. In regard to the possibility of a carcinogenetic effect, Proctor points out that many thousands of patients have been treated and no evidence of such an effect has yet been found in his patients. Most radiologists believe that there is no basis for such a fear.

At the seventieth annual meeting of the American Laryngological Association, Morrison[30] demonstrated a flexible applicator for the application of radium to the lateral pharyngeal areas when introduced through the nose. It has the advantage of being able to reach a larger area by direct application. He lists the indications for this method as "recurring aerotitis, recurring acute catarrhal otitis, prevention of secondary bacterial infection and chronic suppurative otitis media, and for the reduction of hypertrophied lateral bands following tonsillectomy" and the contraindications as "nerve deafness, otosclerosis, atrophic rhinitis and pharyngitis, congenital bursae, recent acute infection and large masses of adenoids." He calls attention to the dangers to the operator as well as the patient. The dangers to the patient may arise from overdosage and the possiblity of late atrophy.

After a brief discussion of the rationale, technique, purposes, contraindications, abuses and dangers of the nasopharyngeal use of the standard monel radium applicator, Morrison[37] describes a new type of flexible radium applicator. It is about the same size as the standard rigid applicator except that the cannula has a little greater diameter. One advantage this applicator has over the standard one is that it enables the operator to reach any lymphoid tissue in the nasopharynx without causing the radium capsule to open. In addition, it permits, for the first time, accurate and comfortable irradiation of the lateral pharyngeal bands and pharyngeal follicles. After the floor of the nose and side of the pharynx are slightly anesthetized, the applicator with its stylet is passed into the nasopharynx through the nose, with the indicating ring turned down. Withdrawal of the stylet completely out of the flexible part permits this part to turn through a right angle so that the radium capsule is in close contact with the lateral band throughout most of its length.

Dow[38] briefly discusses the anatomic, pathologic and physical principles involved in the use of radium for hypertrophic lymphoid tissue in the nasopharynx. The indications for this method of treatment are enumerated and the technique is described. The results obtained in 74 patients treated with radium at the Gill Memorial Eye, Ear and Throat Hospital between February, 1947, and February, 1948, are given. For comparative purposes the series was divided into two groups: *1.* 41 patients in whom the monel metal 50 mg. radium sulphate applicator was placed 8.5 minutes to each side of the nasopharynx, and *2.* 33 patients in whom the same applicator was placed 12 minutes to each side of the nasopharynx. The results indicate that the longer treatment removed lymphoid tissue safely and more effectively.

Spain and Weymuller[39] discuss the value of irradiation of the nasopharynx in the treatment of bronchial asthma in children between one and 15 years of age. They have treated 73 children with bronchial asthma in this way. Of the 43 on whom full data were available, about one-fourth obtained no benefit and about three-fourths were partially or completely relieved of asthmatic symptoms. They point out that asthmatic children with hypertrophied and infected lymphoid tissue are generally sensitive to one or more airborne excitants or foods and this infected tissue seems to act as a focus when bacteria complicate bronchial asthma of allergic origin. It is in these cases that irradiation has proved of benefit. They conclude that "in experienced hands and in selected cases irradiation therapy offers a safe and effective means of combatting infective asthma in childhood."

Fabricant[40] briefly reviews the development by Crowe and associates of the concept that conduction deafness is due to hypertrophy of lymphoid tissue in and around the pharyngeal orifice of the Eustachian tube and its treatment by irradiation; however, he quotes several authorities who are of the opinion that irradiation of hyperplastic lymphoid tissue of the nasopharynx may do more harm than good unless cases are properly selected. Selection of cases for this type of treatment should be based on pathologic findings rather than symptoms. One of the real dangers of irradiation is the possibility of producing radiation damage of tissue, particularly years later. Fabricant cites a study of nasopharyngeal lymphoid tissue and hearing made by Gill on several hundred children. Of 259 children with good hearing not treated and observed for an average of six and one-half years there was a slight gain in hearing acuity for low tones and slight loss for high tones. In a group of children with impaired hearing for high tones only and treated by irradiation of nasopharyngeal lymphoid tissue there was an average gain for low tones similar to the other group and slight additional loss for high tones.

Swanberg[41] reminds us that since 1924 Crowe and associates have been using radon and radium in the nasopharynx to diminish hyperplastic lymphoid tissue. Lymphoid tissue has been proved to be radiosensitive and, therefore, is easily destroyed. Burnam and Crowe devised the original monel metal radium applicator which is now so widely used throughout the country. Swanberg presents his modification of the Burnam-Crowe applicator and claims as new features that: *1.* the applicator is slenderer; *2.* the handle of the applicator is longer and graduated in centimeters; *3*. the radium capsule has a thread hole, can be quickly unscrewed from the introducing handle and is available for usage other than in the nasopharynx; and *4*. there is a dummy applicator for demonstration to the patient. The technique and indications for application of the radium are essentially the same as outlined in all articles on this subject. Swanberg's conclusions are the same as those of Crowe and associates. Except for the presentation of slight changes of questionable merit in the applicator, this entire article represents quotations from Crowe and associates.

In an interesting article containing valuable observations, Bernier[11] discusses the effects of the detonation of the atomic bomb in Japan in 1945. He describes the physical aspects of the bomb burst and roughly divides the casualties into several categories. This paper, however, deals chiefly with those who succumbed to the effects of ionizing radiation. Although considerable observations were made on casualties from lethal doses of ionizing radiation, little information is available on the changes which occurred in the oral regions in this group. According to Beck and Meissner, the terminal picture was that of extreme exhaustion, with blood oozing from mouth and anus, petechial hemorrhages and internal hemorrhages demonstrable only at necropsy. A second group of patients exhibited no changes until between the seventh and thirtieth day after the explosion. The majority of these had received lethal doses of gamma radiation. In these victims hemorrhagic gingivitis usually associated with tonsillitis, pharyngitis and laryngitis developed. In the more severely affected in this group, the lesions, particularly those of the trachea and female genitals, showed severely necrotic ulcerations. The picture was essentially as in agranulocytic angina.

In the third group a less severe form of radiation injury occurred. These patients showed early epilation with pharyngeal angina, which in the majority of cases did not appear until the third or fifth week after bombing. In general the inflammatory changes in the mouth, gingiva and throat were much less severe and reached a climax during the fourth week.

Bernier also discusses the changes in the blood and describes accurately with illustrations the histopathologic changes in the tissue. Sections from the tonsils showed extensive necrosis of epithelium.

SURGERY.

Portmann[43] suggests a translaryngeal approach for operations at the base of the tongue. The various steps in the operation are described and illustrated. Portmann states that the advantages of this approach are the security it offers, the opportunity of observing for a long time the development of the lingual cicatrization and the opportunity of placing tubes of radium, when necessary.

TRAUMA.

Shy and Carmichael[44] report a case of persistent rhythmic contractions of the ipsilateral pharynx, larynx, vocal cord, face and arm in a patient following attempted suicide by projection of an ordinary lead pencil into the skull via the right orbit. A detailed description of the findings on physical examination is given.

BURSA.

Adair[45] describes two cases of pharyngeal bursa associated with cleft palate. He states that only one other similar case has been reported in the literature. His first case was in a boy, 18 months old, in whom the bursa was discovered during an operation for repair of a postalveolar cleft. The second was discovered during examination of a man for a cleft palate.

FOREIGN BODY.

King[46] reports the case of a man, aged 26 years, with a history of recurrent attacks of swelling in the left side of the neck associated with painful swallowing and some fever. Roentgenograms taken during these attacks failed to disclose any evidence of calculi or opaque foreign bodies. These episodes were treated by conservative measures without relief. Fifteen months after onset of symptoms there was still no change in the physical signs, although results of laboratory tests were within normal limits. Injections of iodized oil through the sinus opening in the left tonsillar fossa revealed a sinus tract contiguous with the left submaxillary gland and Wharton's duct. Injection of methylene blue through the same opening revealed free expulsion of the dye at the frenulum of the tongue. Because of these findings operation was undertaken. At the anterior portion of the posterior belly of the digastric muscle a pocket with a diameter of about 1 cm. was found to contain a bristle, like that of a tooth brush. This was removed and the pocket thoroughly curetted. Recovery was without incident.

King states that he has never heard of a case similar to this one and presumes that the bristle had "entered the left peritonsillar fossa and dissected laterally into the neck, setting up an inflammatory process which extended anteriorly to the submaxillary gland and duct and produced sialadenitis and a through and through sinus tract extending from the tonsillar fossa to the frenulum of the tongue."

STENOSIS.

Sanders[47] devised an artificial oropharynx which he successfully used in the surgical treatment of an extensive oropharyngeal stricture in a girl 10 years old. The child had been perfectly normal until three weeks following tonsillectomy and adenoidectomy, when scar tissue began causing difficulty in breathing and swallowing. This tissue was removed at intervals of about two weeks on a number of occasions but each time it would grow back. The child was extremely apprehensive and in poor physical condition as a result of this. Sanders realized that after he removed the scar tissue, he would have to find something that could be anchored in the pharynx to prevent the formation of more scar tissue and at the same time allow no further contraction of the existing cicatrix. Accordingly, with the assistance of a dentist, he devised an artificial oropharynx which provided a usable oropharynx during the period of surgical removal and reconstruction, prevented further contraction of scar tissue and facilitated plastic repair by providing a practical base for application of the skin graft. The patient was perfectly normal at the time of this report, six years postoperatively.

Goodyear[48] reports a method of treating nasopharyngeal atresia which has proved satisfactory in an extremely difficult case. The patient, a man 38 years old, had had 19 previous operations on the nasopharynx for complete nasopharyngeal atresia without relief. The nasopharynx was completely closed by a scarred, hard, fibrous mass. After local injection of procaine hydrochloride a sagittal incision was made and an opening bored with a Thornwald's antrum trephine. An Ingal's gold nasofrontal sinus tube, which had been previously cut to 17 mm. and had horizontal cuts made about the tube and the sections expanded, was inserted with half of a gelatin capsule over the cut edges in order to hold the leaves together. At the time of this report, a year and a half postoperatively, the patient was completely pleased with the result. The patient was given a wire applicator with the end bent at a right angle to use to dislodge any hard particles of food which might obstruct the tube.

Dohlman and Thulin[49] state that pharyngeal stenosis is not an unusual condition and in its milder forms is asymptomatic. Three types are recognized: the most common palatopharyngeal, the glossopharyngeal and a rather rare type of glossopalatinal. In the past the principal cause was syphilis, but in recent years traumatism has been by far the most prevalent cause, especially after tonsillectomy, adenoidectomy, or both. Guillotine removal of tonsils in unskilled hands is especially at fault. Treatment is surgical plus some type of dilatation, and is necessary only if the condition is symptomatic. Palatopharyngeal stenosis is best treated by placing a suture through the bridge and either attaching a lead weight to this, which divides the bridge in several weeks' time, or secondary section. Glossopharyngeal stenosis, which is more difficult to treat, requires plastic methods.

Vaughan[60] reports a modification of a previous technique for the correction of nasopharyngeal stenosis. It consists of passing a stiff, nearly right-angled probe through the opening behind the uvula into the nasopharynx. From the upper limit of the scar attachment, a vertical incision, 2.5 cm. in length was made on each side of the uvula. On each end of the vertical incision, horizontal incisions of about 2.5 cm. were extended laterally. On each side, about 6 mm. below the upper incision, another horizontal incision of the same length was made, thus forming an "E" on one side and a reversed "E" on the other. These four flaps were elevated and retracted after sutures were passed through their inner margins. A suture through the tip of the uvula permitted exposure of the operative field after the soft palate had been drawn forward. The raw surface on the pharyngeal wall posterior to the palate was covered by rotating the lower flap upward; the lower flap was sutured with fine silk. The upper narrow flap on each side was sutured to the raw surface along the posterior border of the mobilized soft palate. Sutures were removed on the eighth day. A dilator the size of the opening and well lubricated with vaseline was passed behind the palate by the patient every day, beginning at the end of the second week and continued for several weeks. From the time of operation the patient was able to breathe freely through the nose.

BENIGN TUMORS.

LeJeune and Steele[61] report a case of angioendothelioma of the hypopharynx which is believed to be the second to be reported in the literature. The patient, a man 45 years old, had had dysphagia for 15 years. On admission, he could swallow no solid foods, was extremely dyspneic and emaciated, and exhibited a change in the quality of his voice. Indirect laryngoscopy revealed a smooth, glistening, grayish mass, about 2.5 cm. by 4 cm. in size, nearly filling the hypopharynx. Low tracheotomy was performed on the day of admission to relieve the dyspnea and two days later an attempt was made to remove the mass but had to be abandoned when apnea developed. The tumor was completely removed at a later date by suspension laryngoscopy, which permitted bimanual operative manipulation and excision. No other method of peroral surgical approach to the lesion would have been as successful as suspension laryngoscopy in this case.

Sambataro[52] reports two cases of cavernous angioma of the oral cavity and pharynx. In the first case, a man 22 years old, the growth extended throughout the entire right tonsillar cavity. The base of the tongue and entire right glossoepiglottic vallecula were extensively involved. The pyriform recess did not appear to be invaded, but the tumor seemed to have surrounded the right lateral border of the epiglottis and occupied the right false vocal cord. The entire growth was removed by four applications of the monopolar electrode. This left a flexible scar and resulted in return of the voice to normal. In the second case the growth appeared as an irregularly lobate, violet colored elevation on the right side of the undersurface of the tongue. Coagulation therapy gave good results. For this reason Sambataro advocates coagulation in the treatment of these types of lesions. Following such treatment the size of the mass is immediately reduced, and as coagulation occurs a narrow halo of whitish color forms around the area of application of the electrode. This helps the operator to know at all times exactly how far the process of coagulation has proceeded.

Bey[53] reports the case of a girl, 16 years old, who had a spheno-occipital chordoma which was unusual in that it felt bony-hard. Almost all tumors of this type previously described have been said to have a "tense jelly-like feel." The patient had had complete nasal obstruction for two and one-half years. Posterior rhinoscopy revealed a large mass attached by a wide base to the roof of the nasopharynx and completely obstructing the postnasal space. The mass looked smooth and round but felt bony-hard. Roentgenograms showed an ovoid, well limited channel in relation to the base of the skull filling the cavity of the nasopharynx; it arose from the basiocciput, basisphenoid and extended forward in relation to the anteroinferior wall of the sphenoid sinuses and laterally almost to the foramen ovale. The diagnosis of fibroma was excluded because of the hard feel of the mass and osteoma was ruled out by the Roentgenograms. After preliminary tracheotomy the tumor was exposed by a left lateral rhinotomy and was removed. Biopsy of tissues from the tumor revealed it to be a chordoma. At the time of Bey's report, two and one-half years postoperatively, there was no sign of recurrence.

Slaughter and DePeyster[34] describe a syndrome produced by neurilemmonas which they believe has never been described before. They report four cases, three of which arose from the vagus and one from the hypoglossal nerve. These cases presented several points in common which Slaughter and DePeyster believe should permit a presumptive diagnosis of neurilemmona of cranial nerve origin. These features are a bulging, firm submucosal tumor in the posterolateral pharyngeal wall, causing dysphagia and later interference with the airway; pulsation of the tumor due to medial displacement of the internal carotid artery and interference with function of the involved nerve. Treatment consisted of extirpation of the mass through an external cervical approach.

Brewer[35] reports a case of a relatively infrequent condition —hypopharyngeal polyp. The patient, a woman aged 55 years, had complained for two years of intermittent attacks of nausea associated with the feeling of a moving lump in the throat. On two occasions a long, thick, red object was regurgitated and hung out of the mouth down to the chin. Indirect laryngoscopy revealed a small, rounded polypoid mass lying in the pyriform sinus. By direct laryngoscopy, about 1.5 cm. of the polyp was teased upward; the mass was seen to be attached by a broad base to the left side of the hypopharynx on its posterior wall. Following removal of the polyp under anesthesia the patient continues to be asymptomatic. The microscopic diagnosis was benign fibromatous polyp.

Wilkerson and Cayce[36] report the case of a girl 19 years old with a hemangioma of the lateral band of the pharynx and nasopharynx, which was removed but recurred in a totally different form. A diagnosis of hemangioma was made preoperatively. Under topical cocaine anesthesia (infiltrating around the mass was deemed undesirable because of hemorrhage) the entire mass, along with the 2 or 3 mm. of apparently normal tissue, was electrocoagulated. The patient returned three months after operation with a pedunculated mass which had three lobules in about the same location as the first tumor. The entire mass was removed, together with a pedicle, and tonsillectomy was done. There had been no recurrence two years after the last operation. Wilkerson and Cayce were able to find in the literature only two other reports of hemangioma arising from and limited to the lateral pharyngeal band.

In a letter to the editor, McFarland[37] adds another case of nasopharyngeal fibroma to the reports in the literature. The patient, aged 14½ years, was found on biopsy to have a highly vascular fibroma in the right nostril which was treated for two years with different sclerosing fluids without changing its size. Because of infection and exophthalmos, it was decided to remove the growth. It was about the size of a tangerine and was attached to the sphenoid body with prolongations into the ethmoidal labyrinth. Recent examination at the time of the report showed no evidence of the growth but there was still slight persisting exophthalmos.

MALIGNANT TUMORS.

A discussion of the surgical treatment of carcinoma of the esophagus must necessarily include lesions involving the pharynx or at least a portion of it. Wookey[38] points out that cancer of the esophagus is much more common in men than in women, about 80 per cent of the cases occurring in men, whereas malignant growths in the lower part of the hypopharynx, known as the retrocricoid area, are almost entirely confined to women. Early diagnosis is the exception, for the distinctive symptom of dysphagia is often late in appearing and in the hypopharyngeal cases may be entirely absent. The first difficulty noted is usually eating solid foods. Hoarseness due to paralysis of a vocal cord and persistent pain in the chest and back are considered serious symptoms, indicating that the growth has penetrated the wall of the esophagus. Wookey urges that a patient who complains of discomfort in swallowing be subjected to a careful thorough examination of the pharynx and esophagus.

Surgical operations devised for the removal of malignant growths in the hypopharynx and mouth of the esophagus should include methods of reconstruction. Lesions of the hypopharynx usually involve the posterior portion of the larynx and in reality are extrinsic carcinomas of the larynx and the larynx must be extirpated.

A complete and detailed description of the surgical procedure used by Wookey is presented. This is an excellent article for those who are interested in this type of major surgical procedure.

From a review of some of the cases of chronic tubal obstruction encountered at the otologic clinic, Perlman[40] concluded that the clinical diagnosis of this condition is based upon a variety of clinical pictures and nasopharyngeal findings as well as of secondary anatomic and functional changes commonly considered as resulting from tubal obstruction. He discusses some of the numerous examinations and tests which have been carried out in an effort to help remove the ambiguity of this condition and gives some clinical impressions obtained from his study. He points out that one of the most clear-cut clinical pictures of chronic tubal obstruction in which therapy can be assessed is in carcinoma of the nasopharynx. Unilateral invasion and occlusion of the tube is often an early complication in these cases. It is true that nasopharyngeal carcinoma is a rare cause of tubal obstruction, but it helps to show that any tissue of the normal tube may cause obstruction, especially in adults. In children lymphoid tissue is the most likely element causing obstruction, but other etiologic factors include inflammation and allergic changes in the mass of secretory glands within the tubes, as well as the fact that transient increases in the size of the lymphoid tissue itself and the glandular element occur with inflammation and allergic reaction and perhaps in response to endocrine stimulation.

Piccione and Foldes[60] discuss the symptoms and diagnosis of lymphoepithelioma and report a fatal case in a woman 42 years old. They emphasize the importance of nasopharyngeal examination in cervical adenopathy or nasal or oral symptoms and early treatment. In the case reported the patient initially noted difficulty in breathing through the nose and had a few nose bleeds. She then began to have headaches, bilateral exophthalmos, failing vision with complete blindness, pronounced nasal obstruction and dysphagia. Permission for biopsy could not be obtained, the patient's condition gradually became worse and she died about one month after admission to the hospital.

In a splendid presentation that should be read before referring patients for irradiation, Jean Tracy[61] describes the various techniques used by the radiologist to obtain desirable radiographs of malignancies of the upper respiratory tract. A brief survey of the anatomy of the nasopharynx, pharynx and larynx and the peculiarities of these structures is presented. The most frequent site in which the common tumors occur is discussed. There follows a discussion of tumors of the nasopharynx, postcricoid region, thyroid gland, epiglottis, vocal cords, larynx and pharynx. The best method to employ is described, and the results obtained are tabulated.

In Part II of this article Annette Lee discusses in a clear and concise manner the various methods of treatment by irradiation for carcinomas of the larynx and pharynx. She states that intrinsic lesions of the larynx may be treated by the Finzi-Harmer fenestration technique using radium or by subcutaneous irradiation. Each method is described in detail with illustrations and with some additional notes on the post-therapeutic care of the patient. Good nursing and nutrition are important and proper attention should be given to patients showing severe reactions to therapy.

Diehl[62] adds to the literature an account of one of the rarer types of nasopharyngeal tumors, a salivary adenocarcinoma. The patient, a man 34 years old, complained chiefly of difficulty in hearing in the left ear. Examination revealed a reddened, fungating mass, extending from the fossa of Rosenmüller inferiorly and involving the left torus tubarius and partially occluding the left Eustachian orifice. The pathologic diagnosis following examination of a specimen obtained by biopsy was carcinoma of an aberrant salivary gland of the left portion of the nasopharynx. The mass was removed and intensive Roentgen radium irradiation was given. The patient had had no further decrease of hearing in the left ear since the original audiogram and one year postoperatively examination shows no evidence of recurrence.

In an excellent article which should be read in its entirety, Morrison and associates[43] present the first published report of the smear technique and cytologic studies for the diagnosis of exfoliating lesions of the nasopharynx. This study was prompted by the use of the smear technique by others for detecting cancer cells in the vaginal and bronchial secretions and by the difficulty in early diagnosis of lesions of the nasopharynx. The normal cell content of the nasopharynx was first determined and as a control the technique was applied also to visible lesions of the mouth, tonsils and larynx. The technique consists of obtaining material from the surface of the nasopharynx, after the nasal mucous membrane has been anesthetized and shrunk. This material is placed on a slide and fixed and stained by the modified method of Papanicolaou and Trout. Diagnosis is dependent upon the finding of malignant cells.

In 85 patients examined there were seven positive smears. There were no false positives and the one false negative (negative smear positive biopsy) was not an exfoliating lesion.

Attention is called to the fact that malignant cells found in the nasopharynx may have come from the nose or sinuses rather than from a lesion in the nasopharynx itself. This procedure is not advocated for routine examination. The importance of differentiating the malignant from the normal cells found in this region is stressed.

Baylin and associates[64] present a Roentgenologic and clinical study of 32 proved cases of nasopharyngeal malignancy, 26 of which were lymphoepitheliomas and six transitional cell carcinomas. Most of these patients sought medical aid because of sinus trouble, aural infection or similar conditions. The lesion is manifested clinically in a variety of ways; neurologic, lymphatic, nasal, ocular and auditory complaints were noted in their patients.

Baylin and co-workers stress the importance of Roentgenographic studies in these cases. Not only lateral views but also special projections, including basal or axial views, stereoscopic studies and accessory views should be made. Of their 32 patients, 25 showed, on careful Roentgenologic study, an abnormality of the nasopharyngeal airway seen either on the lateral skull film or on the mentovertex study. Osseous changes in the skull occurred with such regularity that they are considered almost diagnostic by Baylin and co-workers. Both the primary lesion and the sites of extension and metastases are amenable to irradiation.

In a short but excellent article Munro Black[65] discusses tumors arising in the nasopharynx and enumerates the symptoms produced by them. If the nasopharyngeal tumor is malignant, it may extend and not only infiltrate the surrounding structures at the base of the skull but also interfere with their functions. Further extension of the malignant mass is indicated by gradual involvement of the nerves with resultant palsies of their supplied muscles and by referred pain. Adenoid hypertrophy, nasopharyngeal polypi and fibromas are discussed. The latter type of tumor is admittedly a hazardous type of tumor to attempt to excise. Better results are obtained by interstitial irradiation and multiple recurring diathermy punctures. All varieties of malignant neoplasms may occur in the nasopharynx and 32 per cent of these cases produce cervical adenopathy, 30 per cent nasopharyngeal obstruction, 23 per cent otitic symptoms and 15 per cent neurologic troubles according to Munro Black. The early diagnosis is at times difficult because of the obscurity of the lesion. Biopsy should always be performed as a guide to prognosis and to treatment. Deep Roentgen-ray therapy is the only method that offers a ray of hope; according to Munro Black the five-year survival rate is only 20 per cent in malignant cases. Patients with neurologic symptoms have only a 10 per cent rate of cure and the most radiosensitive lesions reach a survival rate of 40 per cent. It has been my observation that radiosensitive tumors of the nasopharynx give a much higher survival rate than that reported in this article.

Simmons and Ariel[64] present an analysis of 150 consecutive cases of nasopharyngeal cancer in patients treated at the Veterans Administration Hospital, Hines, Ill. They give a brief discussion of the anatomy, emphasizing the lymphatics. Because of the select group in this series, the age, sex and racial incidences are of little value. They found no relation between syphilis, smoking or alcoholic intake and the production of nasopharyngeal cancer.

The gross pathologic condition varied from a small bulge in the nasopharynx to large fungating lesions filling the whole nasopharynx. The most frequent location of the lesions was in Rosenmüller's fossa. In this series epidermoid carcinoma comprised 34.7 per cent, transitional cell carcinoma 30.7 per cent, anaplastic carcinoma 9.3 per cent and the remainder were unclassified. One or more cranial nerves were involved on admission in 29 per cent of the cases. The abducens was most frequently involved, followed by the hypoglossal and the trigeminal.

Eighty-six per cent of the patients had cervical metastases; the upper deep cervical lymph nodes were most frequently involved.

The commonest symptom was swelling of the neck; pain over the distribution of the second division of the Vth nerve was second, and symptoms due to obstruction of the Eustachian tube were third. Gradenigo's syndrome, Trotter's triad and the jugular foramen syndrome were all observed.

Early diagnosis is difficult because there are no early manifestations, and the area involved is extremely inaccessible.

Diagnosis depends upon careful examination of the nasopharynx and biopsy of any suspicious area. Any patient with cervical metastasis in whom no primary lesion has been found should have a careful examination of the nasopharynx. The diagnosis is usually not made by the referring physician and many patients had had surgical treatment without the diagnosis having been made. Roentgenograms were not beneficial in making the diagnosis in this series.

The treatment of nasopharyngeal cancer is exclusively by means of irradiation. The methods and dosage used are described in detail. In 78 determinates, in all of which Roentgen-ray therapy was employed, the five-year survival rate was 9.8 per cent. No cures were obtained in patients with intracranial extension.

Baclesse[67] warns that the prognosis for cancer of the hypopharynx is poor because lesions occurring in this area are usually of an ulcerating and destructive nature. Complications, such as infection, involvement of the cartilages of the larynx and extension add to the difficulty of obtaining cures.

Baclesse divides the hypopharynx into upper and lower divisions and describes each. He attaches much importance to this division, as those lesions occurring in the upper division are more often fungating and bulky, whereas those in the lower division are more often ulcerative and destructive. Among patients at the Curie Foundation, those surviving five years were the ones with lesions in the upper division of the hypopharynx.

Laryngoscopic examination is necessary to determine the origin of the lesion. Radiologic signs of cancer arising in the upper and lower divisions of the hypopharynx are enumerated. Experience has shown that cancer of the lower division of the hypopharynx occurs with greater frequency than in the upper division. Lymph node metastasis increases the gravity of the existing condition and, although such nodes responded to therapy with clinical disappearance in 50 cases out of 186 for a period of six months to one year or more, there was eventually local recurrence.

Baclesse discusses in a thorough manner the technique for administration of radiotherapy employed at the Curie Foundation. Few articles have been published on cancer of the hypopharynx, and fewer still contain anything new. Baclesse suggests proper classification as to location of the lesion and Roentgenologic diagnosis as to location. He further shows that prognosis is good in one and bad in the other. This is a splendid article well worth reading.

Rosenberger[68] gives a brief but excellent discussion of tumors of the nasopharynx. He discusses the juvenile type of fibroma, which is not malignant but can cause serious complications by its erosive qualities. Treatment of this tumor is difficult but *surgical removal preceded by radiation is often* indicated. It may be necessary to split the palate to gain access to the tumor.

Nasopharyngeal malignancy as a result of a wide diversity of tissue types is discussed. *Many types of malignancies are* found in the nasopharynx. Thorough examination of the nasopharynx is stressed and the fact is pointed out that in 50 per cent of cases the first symptom is cervical metastasis, usually appearing just below the mastoid tip. Nasal obstruction, rhinorrhea, sometimes sanguineous, and symptoms of Eustachian tube obstruction are important complaints.

The results of treatment are not good. Surgical measures are usually impossible and the treatment of choice is irradiation. Rosenberger says that Hayes Martin, of Memorial Hospital, reported 25 per cent five-year cures of all types of cases.

Boyce and Bolker[69] point out that, although ocular manifestations of nasopharyngeal tumors are common, only two reports on this subject have appeared in the literature. For this reason they considered it worthwhile to present the results of a survey of 44 cases of nasopharyngeal tumors encountered during the past 15 years at University Hospital in Iowa City. The age and sex distributions and the incidence of types of tumors were similar to those generally reported. Thirty-two per cent of the patients in their series had one or more ocular manifestations at some time during the course of the disease. Most of these were found to have malignant tumors. Multiple ocular manifestations were present in 64 per cent of their patients. Abducens palsy was the commonest ocular complaint, whereas oculomotor and troclear nerve paralyses were noted in fewer cases; four patients had unilateral proptosis. Three typical cases of nasopharyngeal tumors with ocular manifestations are cited. Boyce and Bolker state that the ophthalmologist should be aware of nasopharyngeal tumors since they frequently see these patients first and consequently have an opportunity to discover the lesion early when prompt treatment would be more beneficial.

An editorial[70] by Hollender and Fabricant deplores the failure to recognize nasopharyngeal malignancy until metastatic involvement has taken place. Late recognition is attributed to failure to suspect a lesion in an obscure area, such as the nasopharynx, and to the relative inaccessibility of this area for routine inspection by a nonspecialist. The diagnostic value of Roentgenograms and certain neurologic symptoms, such as headache, pain, diplopia and paralysis of cranial nerves, is emphasized. Roentgen-ray therapy is considered more effective than surgical therapy once metastasis has occurred. Even with such treatment the prognosis is not particularly encouraging.

Grotts[71] reports a case of transitional cell carcinoma of the nasopharynx in a child 29 months old. In addition to the rarity of such tumors, Grotts points out that their occurrence is unusual in the age group between birth and five years. On admission the patient had high fever, was dehydrated and had nasal obstruction. Parenteral antibiotic therapy brought the temperature down to normal, but the nasal obstruction persisted. Manual examination disclosed a mass on the left side of the nasopharynx about the size of a cherry. No pus could be obtained from multiple incisions. Biopsy revealed the tumor to be a transitional cell carcinoma of the pharynx. Deep Roentgen-ray treatment was given, but the patient died 10 weeks after onset of symptoms.

Hung[72] reports a case of carcinosarcoma in a man 28 years old. The patient complained of a sensation of a lump in the throat and slight dysphagia, but no severe symptoms. Pressure down the base of the tongue revealed a globular growth rising from the lobe, leaning toward the left side behind the epiglottis. Laryngoscopy disclosed an egg-sized, elliptic, knobby, coarse tumor closing the hypopharynx and the entrance into the larynx. A specimen obtained at biopsy showed a few cell nests of adenocarcinoma embedded in a stroma rich in cells. Because the neoplasm was thought to be most likely a malignant and bleeding one, the tumor was removed by the external route. Because of its large size it had to be excised in pieces. Its origin was not determined until the thirteenth postoperative day when endoscopy revealed that the tumor arose subepithelially with a narrow pedicle from the left pyriform sinus and the left lateral posterior wall of the hypopharynx. Radium irradiation perorally was begun on the eighteenth postoperative day and the patient left the hospital on the thirty-seventh postoperative day for family reasons. Hung believes that this patient may be the youngest patient with a large hypopharyngeal carcinosarcoma recorded in the literature. A discussion of the problem of carcinosarcoma, particularly those of hypopharyngeal origin, is included.

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THE RESEARCH STUDY CLUB OF LOS ANGELES.

The Twentieth Annual Midwinter Postgraduate Clinical Convention in Ophthalmology and Otolaryngology will be held at Los Angeles, Calif., Jan. 15-26, 1951.

The guest speakers for the convention will include, for the Eye—Dr. Derrick Vail and Dr. Irving Puntenny, both of Chicago, Ill., Dr. Frederick C. Cordes, of San Francisco, Calif., and Dr. Meyer Wiener, of Coronado, Calif. In addition, the resident speakers for the Eye will be Dr. Harold F. Whalman, Dr. S. Rodman Irvine, Dr. John A. Bullis, Dr. Warren A. Wilson and Dr. Carrol L. Weeks, all of Los Angeles, Calif.

Dr. Wiener, emeritus professor of clinical ophthalmology, Washington University School of Medicine, St. Louis, Mo., will present his usual course in Surgery of the Eye. Because of the difficulty in handling a larger class for practical demonstration on animal eyes, this special course by Dr. Wiener and associates will be limited to 24 students. Applications will be accepted in the order in which they are received. This course will start Friday afternoon, Jan. 19, and continue Saturday afternoon and all day Sunday. It will consist of a short explanation of the procedure to be followed, with demonstration on animal eyes by the instructors, after which the student himself will perform under supervision. A list of required instruments will be mailed to applicants.

For the Ear, Nose and Throat the guest speakers will include Dr. Louis H. Clerf, of Philadelphia, Pa., and Dr. James H. Maxwell, of Ann Arbor, Mich. Other lectures and instruction courses will be given by Dr. Wilford H. Belknap, of Portland, on Office Procedures; Dr. J. C. Almy Harding, of San Diego, Calif., on Nutrition; Dr. Isaac H. Jones, of Los Angeles, Calif., on Vestibular Problems; Dr. Cyril Courville, of Los Angeles, Calif., on Intracranial Complications from Ear, Nose and Throat Infections; Dr. Gilbert Roy Owen and Dr. Ray Alden Carter and staff, of Los Angeles, Calif., on X-ray Problems; Dr. George S. Sharp, of Pasadena, Calif., on Tumors in Ear, Nose and Throat; Dr. Herman Semenov, of

Beverly Hills, Calif., on Sinusitis Problems; Dr. Howard P. House, of Los Angeles, Calif., on Ear Surgery; Dr. H. James Hara, of Los Angeles, Calif., on Blood Dyscrasias in Ear, Nose and Throat; Dr. Kenneth C. Brandenburg, of Long Beach, Calif., on Psychosomatic Ear, Nose and Throat; Dr. Gilbert J. Roberts, of Pomona, Calif., on Nasal Fractures.

Write for reservations at an early date to H. M. Nickerson, manager of the Elks Club, Parkview at Sixth Street, Los Angeles 5, Calif. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

Each applicant must be a member of his own local medical society *in order to become eligible* for attendance at the course. Send your fee to Dr. Pierre Violé, treasurer, 1930 Wilshire Boulevard, Los Angeles 5, Calif. If anything prevents your attendance, this fee will be returned to you.

The first week will be devoted to the Eye—from Monday to Saturday, Jan. 15 through Jan. 20. The Ear, Nose and Throat week will be from Friday to Friday, Jan. 19 through Jan. 26. Those of us who confine our work to only one of the specialties may complete either subject in one week.

The fee for the Clinical Course is $75.00; the fee for the Special Course by Dr. Wiener is $50.00.

TEMPLE UNIVERSITY COURSE.

Course in Laryngology and Laryngeal Surgery to be given at Temple University School of Medicine, Nov. 6 to 17, 1950. Fee, $250.00. Further information and application blanks sent on request. Dr. Chevalier L. Jackson, 3401 North Broad Street, Philadelphia 40, Pa.

NEW HEARING AID FEATURES WORLD'S TINIEST RECEIVER — OFFERS WIDEST FREQUENCY RANGE, MORE OVERTONES.

A new hearing aid, the Audivox Super 67, featuring the world's tiniest receiver, only a trifle larger in diameter than an ordinary button on a man's shirt, now offers hard-of-hearing patients a new range of higher frequency tones. No other hearing aid receiver known to Audivox hearing experts comes even close to being as tiny as the new Super 67 receiver.

Based on Bell Telephone Laboratories designs, the Audivox 67 is a product of Audivox, Inc., successor to the Western Electric Hearing Aid Division.

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Many physicians, accustomed to patients' objections to wearing an aid because of the conspicuousness of the receiver, will find that the reduced size of the Audivox Super 67 receiver helps *overcome this psychological resistance.*

The tiny size and improved performance of the receiver is made possible by the employment of a "double magnetic" technique of sound reproduction. Entirely new in hearing aid receivers, this technique represents such an advance that the remarkable reduction in size does not result in a loss of efficiency, but actually greatly increases the frequency range.

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The Audivox Super 67 weighs only 6 ounces complete with batteries, and measures 3.5x2.5x0.9 inches.

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Depending upon the receiver used, the output of the Audivox Super 67 varies between 119 and 136 db to give more power than most hard-of-hearing persons require.

The University of Toronto, Faculty of Medicine, announces a combined Refresher Course in Ophthalmology and Otolaryngology during the week of Jan. 29 to Feb. 3, 1951. Dr. Phillips S. Thygeson, University of California School of Medicine, and Dr. Charles Iliff, Johns Hopkins University, will be guest speakers in Ophthalmology. Dr. LeRoy Schall, Harvard University, and Dr. G. E. Tremble, McGill University, will be guest speakers in Otolaryngology. There will be surgical and medical clinics in these two subjects in addition to lectures by members of the faculty.

The course will be given for a minimum of 10 students and a maximum of 25 students. Application may be made to the Dean of the Faculty of Medicine not later than Nov. 30, 1950.

OCTOBER 1, 1950

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM; Model M-35; Model P-15.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co. Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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THE
LARYNGOSCOPE.

VOL. LX NOVEMBER, 1950. No. 11

OFFICE PROCEDURE IN HEARING EVALUATION. A PRACTICAL APPROACH.*

LEROY L. SAWYER, M.D.,
Washington, D. C.

In the past 20 years and particularly during the past seven years so many hearing tests have been suggested and conducted, and so much research on hearing tests has been carried out, that the otologist may well feel confused. He may ask, "Which of these tests is the most reliable?" If in private practice, he may inquire, "Which test is not only reliable but also practical from the standpoint of equipment and time consumption?"

The clinical investigation presented in this paper was undertaken, primarily, for the purpose of determining the essential tests and minimum physical equipment required in private practice for a reliable evaluation of hearing loss. Admittedly, it is with considerable trepidation that the writer approaches this exhaustive subject, to which valuable contributions have been made by many eminent men, including Carhart, Davis, Fowler, Guild, Hoople, Mueller, Reger, Silverman, Walsh and Westlake. It may be that some phases of the test proposed will appear somewhat unscientific, but it should be borne in mind that a concerted effort has been made to arrive at a practical method of testing and evaluation.

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*Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1950.

HISTORICAL REVIEW OF TESTS OF HEARING.

The first method used to measure auditive acuity was the human voice, but this method presented limitations because of variations inherent in the voice and speech, and because of the lack of an accurate system of measurement.

Early examiners turned to the use of the watch, the coin click and the tuning fork as practical mechanical devices for producing uniform sounds. The development of tests with tuning forks is particularly interesting, and will be described further on in this paper. All these instruments proved to have drawbacks due to variability. As stated by Hays, "No two instruments, no two tuning forks, no two watches will ever be alike, so that there is no value in comparing the hearing of an individual patient when a test is taken with two different instruments, and by two different men with different watches and tuning forks.*

The next instrument used to measure auditive acuity was the so-called acoumeter, which was first suggested by Wolken.[37] This was a simple device with sound produced by hitting an oaken hammer against a fir plaque at a determined angle. The patient closed his eyes and raised his finger each time he heard the sound. From 1864 several different types of acoumeters were devised. Conta,[6] attempting to estimate how many seconds a sound is heard, devised an instrument making use of a tuning fork as the source of sound. The tuning fork was vibrated by hitting it on the knee, and then its stem was inserted into a rubber tube, which was introduced into the ear. Magnus[25] in his instrument used a tuning fork which was fixed to a table and struck with an ivory ball, always with equal force. At the end of the tuning fork was placed a kind of acoustic horn, in which the sound waves were gathered and projected into a tube. Kessel[20] fabricated an apparatus with the mechanism of a music box. In Politzer's[28] instrument a cylinder encased in rubber was struck with a small hammer.

In 1878, Hartmann[13] described an instrument making use of the recently invented telephone receiver, the tuning fork and controlled electric current. This apparatus, however, was too intricate to be generally accepted. At about the same time, Hughes[17] presented an instrument to the Royal Society which consisted of: *1.* the new induction currents balance; *2.* microphone, with a clock as a source of sound; *3.* electric sonometer, or absolute sound measurer, and *4.* a receiving telephone and three elements of Daniell's battery.* It was in the work of these two men that the modern audiometer with sound produced electrically had its genesis. Shortly after Hughes introduced his instrument, Richardson[30] proposed its use for measuring the effect of disease, treatment and atmospheric pressure on the acuity of hearing.

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*Hays, H.: An Improved Audiometer. The Laryngoscope, 23:866, 1913.

The instruments devised by Hartmann and Hughes received recognition both in Amerca and Europe, and in the years following their introduction, literature contains many descriptions of audiometers with variations and modifications. Different sources of sound were used, and attempts made to overcome the limitations of the earlier devices, due to their cumbersomeness, complicated construction and restricted range of sound production. Instruments were described by Körting,[21] in 1879; by Lacharrière[23] and by Boudet de Paris,[3] in 1882; by Urbantschitsch,[34] in 1884; by Cozzolino,[7] in 1885; and by Cheval,[6] in 1890. In 1880, Maillard[26] replaced the clock and microphone used in Hughes' instrument by a small Gaiffe battery equipped with an interrupter which produced the sound. Variations suggested by Jacobson,[19] in 1885, consisted of a second induction coil for controlling the volume of sound produced, and the use of two rubber tubes for transmission of the sound to the ears. An audiometer was described by Henry,[18] in 1896. Bezold[1] devised a continuous tone series of tuning forks to provide a wider range of sound. In 1904, Reid[29] described another audiometer. An improvement in the audiometer was suggested by Faught,[10] in 1911, which permitted measuring the acuity of hearing in either ear to the exclusion of the other, and the instrument was so controlled that the patient was unable to know either the intensity of sound or which ear was being tested. Hays,[14] in 1913, devised an audiometer using the watch and tuning fork, whereby the hearing distance of a patient would vary less than 0.5 cm.

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*Hughes, D. E.: Nature, 20:78, 1879.

In 1914, Stefanini[33] made an important contribution to the development of the audiometer in introducing an electric generator capable of a wider range of frequencies. It remained for Bunch in collaboration with Dean,[9] in 1919, to apply this type of generator to the audiometer. The new pitch range audiometer made it possible to produce tones between 30 and 10,000 cps. in a continuous sweep through the entire range of pitch and intensity. It produced tone relatively pure; it provided for varying extremes of intensity; and it permitted accurate measurement both as to pitch and intensity of tones. The apparatus could be operated quickly and easily and had practical application for the measurement of conventional hearing tests. Although the instrument was applied to clinical otology, it was not available commercially.

The next important step in the development of the modern audiometer was the utilization of the vacuum tube by Minton and Wilson[27] and by Guttman,[12] in the year 1921. The following year, as the result of research by the American Telephone and Telegraph Co., the Bell Telephone Co. and the Western Electric Co., the Western Electric 1-A Audiometer utilizing pure tones as the stimulus was produced. It was presented to otologists by Fowler and Wegel,[11] in 1922. Because of the cost of the instrument, it was not generally adopted, and the Western Electric Co. went on to devise the less expensive audiometer, the 2-A.

Since 1922, many improved models of audiometers have been developed and placed on the market. Rapid strides have been made particularly since 1945, and there are now available several audiometers that fulfill all the requirements for the evaluation of hearing loss.

The utilization of recorded words on a wax cyclinder, in place of pure tones as the source of sound, was first advocated by W. S. Bryant,[1] in 1904. The test consisted of selected monosyllables recorded at a constant level. The phonograph was enclosed in a soundproof box from which the acoustic signals were conveyed to the listener's ear by a tube. This tube was equipped with a valve which, by regulating the diameter of the tube, controlled the intensity of the signal. Thus it was possible to express hearing loss in terms of different degrees of valve openings at the threshold of normal and defective ears. Due, probably, to the relative crudeness of the phonographic equipment of that day, the test was not widely used.

In 1926, the Bell Telephone Co., in cooperation with the American Federation of Organizations of the Hard of Hearing, developed the first widely used recorded auditory test for determination of hearing loss with speech. This test consisted of spoken digits and was known as 4-A; today it is known as 4-C. Following research at the Psycho-Acoustic Laboratory of Harvard University, recorded auditory tests were developed for measuring hearing loss with speech. With but slight modifications, these tests are accepted today as the most reliable recorded auditory tests.

Tuning Fork Tests. In the sixteenth century, Ingrassia[18] called attention to the fact that it was possible to hear a vibrating tuning fork when it was held against the teeth. In this fashion the phenomenon of bone conduction was observed. It was not until about three centuries later that the classical tuning fork tests were developed which formed the basis of differential diagnosis of conductive and perceptive deafness.

The first of these tests was described by Weber,[35] in 1834, as follows: "If both ears are closed by the hands, one's own voice is better heard than with open ears. If one ear is closed, one's own voice is better heard by the closed ear. If a tuning fork is pressed against the teeth and the mouth closed as firmly as possible and the ears are closed by the fingers, the tuning fork is better heard than with open ears; however, when the meatus of one ear is closed and the other one is open, the sound is stronger heard in the closed than in the open one." Weber also noted that a deaf person may hear a tuning fork pressed against the skull better in the ear with the greater loss of hearing.

Ten years later, Bonnafont[2] applied Weber's test clinically for diagnostic purposes. Using a series of tuning forks with a wider range of sound than that of the ordinary tuning fork, he sought to diagnose accurately certain types of deafness and to classify those which were incurable with medication.

The second test was suggested by Rinne,[31] in 1855, and was based on the practice of holding the tuning fork first against the upper incisors and then near the external auditory meatus, making it possible to compare air and bone conduction.

In 1885, Schwabach[32] described a bone conduction test in which a tuning fork was placed against the vertex. To differentiate between conductive and perceptive deafness, he considered it of importance to determine how long a deaf person heard the tuning fork, and compare the time with the time it was heard by a person with normal hearing.

Clinical application of bone conduction tests was made by Lucae[24] and by Bezold.[1] The latter made use of a series of clamped tuning forks, which gave a greater uniformity in intensity of tone than other forms of apparatus in use. In slight degrees of ankylosis of the stapes in unilateral sclerosis, he noted that Rinne's test showed a shortened positive or negative result, even in the opposite ear, despite the fact that the hearing for speech in this side was normal or nearly so. In exudative processes and swollen lesions of the middle ear, Rinne's test was found to be positive, although shortened, despite a relatively high degree of deafness for the voice. When one ear was very deaf and the other about normal, there was a decidedly positive result with Rinne's test. After traumatic ruptures of the tympanic membrane, Rinne's test was positive, but shortened.

Quantitative tuning fork tests were developed so that the acuity of hearing could be determined over a wide range. Politzer[28] determined the frequency of tuning forks from 32 d.v. to 4,096 d.v., and it is interesting to note that many manufacturers have recognized these numerical values.

INVESTIGATIONS.

Two investigations were carried out: the first to determine the attenuation effect of headphones and rubber plaster mixing bowls; the second, to evaluate the reliability of certain tuning fork tests, which had been in question for years. Both investigations were conducted in a sound treated room with an ambient noise level of less than 24 db., when measured with a General Radio Co. noise level meter, Type 759-B.[*]

Investigation with Headphones. The matched headphones of a headset were encased in firm rubber and tested for the attenuation effect on spondee words in the free field, on the basis of normal threshold as determined by testing a group of persons. Observations in this test indicated that attenuation for high frequency in the order of 1,000 cps. and above was greater than for frequency below this figure. With this in mind, the attenuation effect of the headphones on PAL noise and on sawtooth noise was investigated. The PAL noise consists of thermal noise upon which is superimposed static that is composed of more high tones than low tones. Sawtooth noise is predominate in vibrations close to 128 cps. or below. First, the attenuation effect was determined when the headphones were clamped tightly over the ears, and second, when held in place by a regular headset.

A similar investigation was made with the headphones encased in sponge rubber.

Finally, rubber plaster mixing bowls, clamped over the ears so as to produce a slight vacuum, were tested for the effect on spondee words in the free field, on PAL noise and on sawtooth noise.

The findings of the investigation are shown in Chart 1. In comparing the attenuation value of headphones, encased in firm rubber and not clamped over the ears, with those encased in sponge rubber and not clamped, for spondee words on the basis of normal threshold tests, the former had an advantage

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[*] The writer wishes to express his appreciation to Dr. Aram Glorig, Jr., and to Mr. Francis L. Sonday for their cooperation, and for permitting the use of the facilities at Forest Glen Aural Rehabilitation Center for these investigations.

CHART 1.

ATTENUATION VALUE OF HEADPHONES AND RUBBER PLASTER MIXING BOWLS.

	Position of Headphones	Spondee Words	PAL Noise	Sawtooth Noise
Two matched headphones encased in firm rubber	Clamped tightly over ears	25 db.	30 db.	14 db.
	Fitted with regular headset	20 db.	20 db.	4 db.
Two matched headphones encased in sponge rubber	Clamped tightly over ears	20 db.	14 db.	8 db.
	Fitted with regular headset	15 db.	4 db.	0 db.
Rubber plaster mixing bowls	Clamped tightly over ears so as to produce a slight vacuum	10-20 db.	28 db.	8 db.

of 5 db. over the latter. The attenuation effect of headphones encased in firm rubber, when not clamped tightly, was 20 db. for PAL noise, whereas they gave only 4 db. of attenuation for sawtooth noise. Rubber plaster mixing bowls clamped tightly so as to produce a slight vacuum had an attenuation effect of from 10 to 20 db. for spondee words, 28 db. for PAL noise and 8 db. for sawtooth noise.

Investigation with Tuning Forks. Three different tests were made in the effort to evaluate tuning forks in hearing tests. Six tuning forks were used in the investigation: two 512 magnesium, one 512 steel, two 1,024 magnesium, and one 1,024 steel.

The first test was performed with the intent of showing the intensity of sound transmitted from the stem of different tuning forks, when they were activated by different methods. Chart 2 shows the variation in volume of sound produced when the tuning forks were hit with a rubber percussion hammer, when they were hit on the knee, and when pinched. The tuning forks were first hit or pinched very hard, and then with what was considered average force. All readings were taken with the stem of the tuning fork balanced on the microphone of the noise level meter, using the tester's finger as a base.

CHART 2.

RESULTS OF DIFFERENT METHODS OF ACTIVATING TUNING FORKS' MEASUREMENTS TAKEN WITH TUNING FORKS BALANCED ON TESTER'S FINGER RESTING ON MICROPHONE.

Tuning Forks	Rubber Hammer		Hitting Knee		Pinch	
	HB	AB	HB	AB	HB	AB
1. 512 mg.	70	65	65	60	50	45
2. 512 mg.	68	65	65	60	50	45
3. 512 steel	65	60	60	55	55	50
1. 1,024 mg.	65	60	60	55	50	45
2. 1,024 mg.	65	60	60	55	50	45
3. 1,024 steel	55	50	50	45	45	40

All readings taken in decibels of intensity as measured on GRC meter. Type 759-B, weighting curve set at C. HB = hard blow; AB = average blow.

In the tests with the magnesium tuning forks of 512 cps. there was a variance in intensity of sound from a maximum of 70 db. to a minimum of 45 db. Steel tuning forks of 512 cps. showed a variation from a maximum of 65 db. to a minimum of 50 db. The 1.024 magnesium tuning forks showed a variation from a maximum of 65 db. to a minimum of 45 db. The 1,024 steel tuning forks showed a variation from a maximum of 55 db. to a minimum of 40 db.

The second test was made for the purpose of comparing the volume of sound emitted by tuning forks when they were activated by a rubber percussion hammer, by being hit on the knee, and by pinching, and then held one-half inch from the microphone. As shown in Chart 3, with the three different methods of activation, there was a maximum difference of 25 db. in the intensity of sound produced when the 512 magnesium tuning forks were tested, a maximum difference of 20 db. when the 512 steel tuning forks were tested, a maximum difference of 25 db. when the 1,024 magnesium tuning forks were tested, and a maximum difference of 15 db. when the 1,024 steel tuning forks were tested.

The third test was performed in the endeavor to compare the intensity of sound produced when the tuning forks were held in three different positions: *1.* when the stem of the tuning fork was balanced on the tester's finger as it rested on

CHART 3.

RESULTS OF DIFFERENT METHODS OF ACTIVATING TUNING FORKS' MEASUREMENTS TAKEN WITH TUNING FORKS HELD ON EDGE, ONE PRONG ABOVE THE OTHER, ONE-HALF INCH FROM MICROPHONE.

Tuning Forks	Rubber Hammer		Hitting Knee		Pinch	
	HB	AB	HB	AB	HB	AB
1. 512 mg.	100	90	90	85	80	75
2. 512 mg.	100	90	90	85	80	75
3. 512 steel	85	80	80	75	70	65
1. 1,024 mg.	100	95	95	90	80	75
2. 1,024 mg.	98	93	95	80	80	75
3. 1,024 steel	80	75	80	75	70	65

All readings taken in decibels of intensity as measured on GRC meter, Type 759-B, weighting curve set at C. HB = hard blow; AB = average blow.

the microphone; *2.* when the tuning fork was held on edge one-half inch above the microphone; and *3.* when the tuning fork was held on its side with the prongs parallel, one-half inch from the microphone. In this test all tuning forks were activated with a rubber percussion hammer.

As shown in Chart 4, there was a considerable variation in the intensity of sound produced when the tuning forks were

CHART 4.

RESULTS OF TESTS WHEN TUNING FORKS ACTIVATED BY A RUBBER PERCUSSION HAMMER AND HELD IN DIFFERENT POSITIONS.

Tuning Forks	Balanced with Finger on Microphone		Held on Edge, ½" from Microphone		Held on Side, ½" from Microphone	
	HB	AB	HB	AB	HB	AB
1. 512 mg.	70	65	100	95	92	85
2. 512 mg.	68	65	100	95	92	85
3. 512 steel	65	60	85	80	85	80
1. 1,024 mg.	70	65	100	95	100	95
2. 1,024 mg.	70	65	100	95	100	95
3. 1,024 steel	60	55	85	75	80	70

All readings taken in decibels of intensity as measured on GRC meter, Type 759-B, weighting curve set at C. HB = hard blow; AB = average blow.

held in the three different positions. The 512 magnesium tuning forks when balanced produced from 70 to 65 db. of intensity of sound; when held on edge, from 100 to 95 db.; and when held on the side, from 92 db. to 85 db. There was, therefore, a maximum difference of 35 db. in intensity of sound produced when the tuning forks were held in the three different positions. In a comparison of the volume of sound emitted when the 512 magnesium tuning forks were held on edge and on the side, there was a difference of from 8 to 10 db. in favor of the position on edge.

The maximum difference in intensity of sound produced in testing the 512 steel tuning forks was 25 db.; in testing the 1,024 magnesium tuning forks, 35 db.; and in testing the 1,024 steel tuning forks, 30 db.

The decrement of the tuning forks was measured. It was found that when the tuning forks were activated with a force as uniform as possible, there was a variance of from 0.66 db. a second to 1.75 db. a second. These figures closely approximate those established by Lierle and Reger.[21] The decrement of the steel tuning forks was found to be practically twice as fast as that of the magnesium tuning forks. This was due to the fact that tuning forks have different physical properties.

PRESENTATION OF HEARING TEST.

The clinical work underlying this test extended over a period of 18 months, but the majority of patients were tested for hearing loss in the period from July 1, 1948, to July 1, 1949. A total of 105 cases were studied. The series does not include any patient under eight years of age, or patients with language difficulty, or mental cases. Thirty-five of the patients were males; 70 were females. The age of the patients ranged from eight to 80 years.

Conditions Under Which the Tests Were Made. All tests were made in the writer's office, which is located in a modern office building. The room was 8 feet by 12 feet in size, and was an inside, air-conditioned room without windows. Two walls, 6 and 8 inches thick, respectively, separated it from the hall corridor. The ceiling and walls were treated with acoustical plaster and covered with sound absorptive material. The doors were covered with sound absorptive material, and the jambs with strips of felt. The floor was heavily carpeted. In a room thus treated, the ambient noise level, when measured with the General Radio Co. noise level meter, Type 759-B, was 37— db. This reading was determined by averaging the maximum and minimum readings for three settings on five different occasions (see Chart 5).

CHART 5.

DETERMINATION OF AMBIENT NOISE LEVEL OF ROOM USED FOR CLINICAL WORK.*

Sound Level Range	Weighting Curve	Readings		Ambient Noise Level of Room†
		Max.	Min.	
24- 55 db.	A (40 db.)	38 db.	28 db.	37— db.
55- 85 db.	B (70 db.)	37 db.	34 db.	
85-140 db.	C (equal response over entire range)	47 db.	43 db.	

*All readings made on GRC sound level meter, Type 759-B.

†The average was obtained by taking the average of the maximum and minimum readings.

When tests are carried out in a room with an ambient noise level of 37— db. and headphones are used that produce 20 db. of attenuation. the ears are actually being tested in an ambient noise level of 17 db.

A modern audiometer and two-speed turntable were used in making all tests. The audiometer was equipped with a microphone, two matched headphones encased in firm rubber, a bone conduction receiver, a high fidelity playback system provided with a trimmer for the reproduction of recordings, and a zero-power level meter. The audiometer was also provided with a calibrated white noise for masking, and another available masker was an air jet. The entire system was equipped with a silent interrupter switch.

Before any tests were made, the audiometer was calibrated for normal speech and pure tone levels under the conditions which were to be used in making the tests. This was accomplished in the following manner: Ten persons whose hearing was known to be normal were tested for air conduction and bone conduction, using pure tones, spondee words (Auditory Test 9), the PB 50-word lists, and connected discourse. The group included three children of 12, 13 and 14 years of age; three college students of 19, 20 and 21 years of age; and four adults of 35, 38, 40 and 45 years of age. The average for each of the tests was taken as normal. Throughout the investigation the corrective factor was used.

Although 10 persons, that is, 20 ears, with normal hearing were used to standardize the equipment in this particular study, it is the writer's opinion that four persons, that is, eight ears, are sufficient. Each six months the system should be recalibrated, using four persons, that is, eight ears.

Method of Procedure. A complete history was taken of each case, including the age, sex, marital status, childhood diseases, past illnesses, and the most recent serious illness. Information relative to the hearing loss was obtained by questioning the patient, or parent, as follows:

1. Is the hearing constant or does it vary?
2. Is the hearing better in a noisy or quiet place?
3. Is there trouble hearing at church, at the movies, on public conveyances, in groups, or on the telephone? Name in the order in which hearing is most troublesome.
4. Is the hearing average, better, or worse today?
5. How long since the last upper respiratory infection?
6. Is there any family history of deafness?
7. When and under what conditions was the hearing loss first noticed?
8. Do you know of any possible cause of hearing loss?
9. Has tinnitus, vertigo, dizziness or allergy been experienced?
10. What was the number of pregnancies and was the hearing affected?

When the history of the case had been obtained, a complete ear, nose and throat examination was made. This was followed, if indicated, by a complete physical examination, blood tests and Roentgenographic examination.

Before any tests were made for either pure tones or speech reception, the patient was first allowed to hear the pure tones and to see a group of words similar to those to be used. Detailed instructions were given as to what to expect during the test. This familiarizing the patient with the procedure is felt to be of inestimable value.

Pure Tone Test: The pure tones air conduction test was made by taking the average of the readings for the following three approaches to threshold: *1.* the threshold was approached from above in 5 db. steps; *2.* the threshold was approached from below in 5 db. steps; and *3.* the threshold was approached from below in 5 db. steps, and at each step the tone was interrupted at the rate of once a second. All air conduction tests started at 1,024 cps. and went first up the scale to 8,192 cps., and then down the scale from 512 cps. to 128 cps. It was observed that the findings for the range of 512, 1,024 and 2,048 cps. did not vary in all three methods by more than 2 db.; however, in the tones of 4,096 cps. and 8,192 cps., when the threshold was approached from below and the tones were interrupted, it was observed that the threshold was lowered by an average of 5 db. at 8,192 cps., and from 3 to 5 db. at 4,096 cps. It was also observed that when the threshold was approached from below threshold in tones of 256 cps. and 128 cps., the threshold was raised by 5 db. In perceptive deafness, this lowering of from 3 to 5 db. in the higher tones was consistently present. In conductive deafness, the 3 to 5 db. of elevation in threshold in tones below 512 cps. was consistently present.

Readings for bone conduction were taken for 512, 1,024, 2,048 and 4,096 cps. It was noted that when pressure was made on the bone conduction receiver, a lowering of 5 db. could be elicited. A similar lowering was elicited when a regular headset was used, and a piece of sponge rubber, two inches thick, placed under the headset on the opposite side of the head, simulating the pressure on the bone conduction receiver that would be produced with a head of larger size. In all bone conduction tests, masking slightly above the threshold of the ear not being tested was used.

Speech Reception Tests: Auditory Test 9 was used for threshold determination for speech. This test consists of a total of 84 spondaic words, which are words of two syllables equally stressed, such as hotdog, railroad and cookbook. Each record of 42 words is in a different scrambling. The lists are recorded in groups of six words progressively lowered by 4 db. steps. The threshold reading for this test is the level at which a patient can repeat correctly 50 per cent of the words. Hudgins, *et al.*,[14] have worked out the method of determining this level. It is of the utmost importance in using Auditory Test 9 that the attenuation is set high enough so that a patient can repeat correctly all of the first six words.

In order to test discrimination, the PB 50-word lists, which were developed at the Psycho-Acoustic Laboratory and recorded in the Technisonic Laboratory at Central Institute for the Deaf, were used. Each list contains 50 words, all of which are monosyllables, phonetically balanced, and which represent proportionately the sounds in the English language as it is spoken. The settings used were 25 db. above the threshold as determined by spondee words, and the maximum intensity tolerated by the patient. It was felt that the setting 25 db. above the threshold as determined by spondee words was best for diagnostic purposes. Seldom would a patient tolerate a maximum intensity of 120 db. In perceptive deafness, as the attenuation was increased above threshold, the score for the PB 50-word lists did not increase, but rather it had a tendency to fall, whereas in conductive deafness, discrimination rose. In conductive deafness, the PB reading for prognostic value was taken at the maximum level; in perceptive deafness it was taken at the maximum comfortable level.

The PB 50-word lists were used both with and without a carrier phrase. It was observed that the carrier phrase, "You will say," had the distinct advantage in young children and in the very old of alerting the patient, and keeping his attention focused on the problem at hand. Curiously enough, a selected few of the more intelligent patients thought that the carrier phrase was monotonous.

A connected discourse test was made, utilizing records of a speech by Fulton Lewis, Jr., and of some recent broadcasts over the NBC System. The most comfortable level for connected discourse was found to be 12 db. above the threshold at which the patient could, with effort, understand each word.

From the readings of Auditory Test 9 and the PB 50-word lists, it was possible by using Davis' chart, the Social Adequacy Index (SAI) for Hearing, to quantitatively evaluate the hearing loss, and to predict just how much improvement might be expected with a hearing aid, or from auditory training, or from a successful fenestration operation.

COMMENT.

A sound treated room is absolutely essential in detecting early hearing loss. The ambient noise level should not be above 45 db. when measured with a weighting curve set at C (equal response over entire range). It is true that the sound level meter does not react just as the human ear, but, nevertheless, it is one of the most practical methods for measuring sound or noise. It is a device, not for measuring frequency and pitch, but for measuring the amplitude of rapid alterations in air pressure. When such alterations occur within a given range of frequency, the ear interprets them as sound or noise. All findings are, therefore, subjective. With a sound level meter, on the other hand, all measurements are objective, since they are unaffected by the many human variables, such as prejudices, likes or dislikes, which enter into any sort of human estimates of loudness.

Charts 6, 7, 8 and 9 show very clearly the importance of using a sound treated room in evaluating early hearing loss.

From an investigation of the attenuation effect of headphones, it was found that with properly constructed and well fitted headphones, it is possible to lower the ambient level of

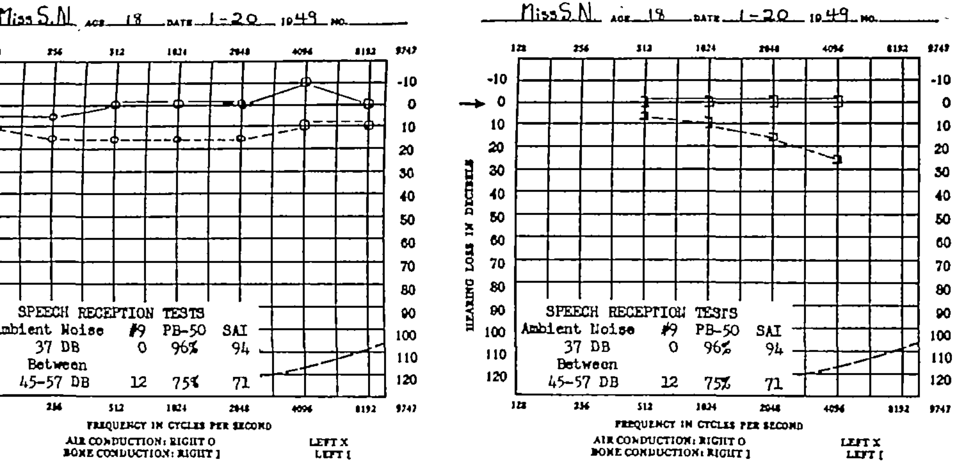
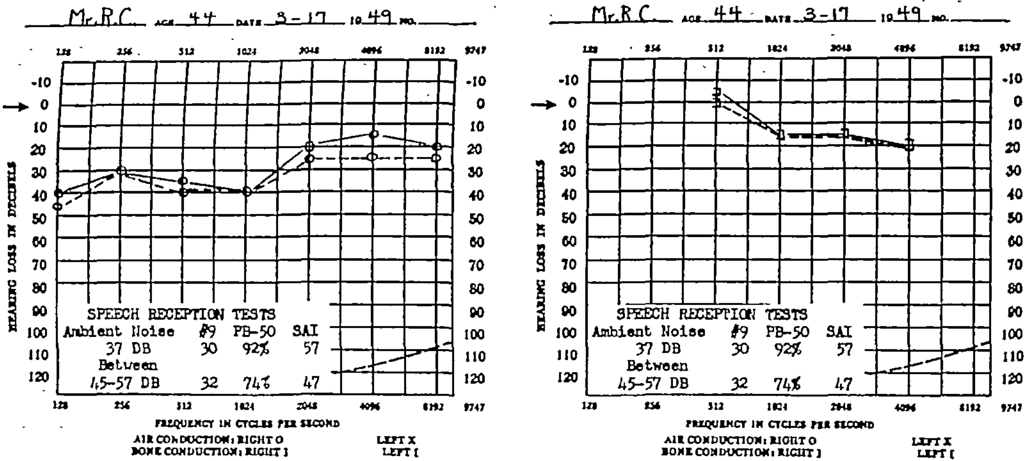


Chart 6. Case 1. S. N. Effects of ambient noise in testing a patient with normal hearing. Solid line indicates tests in om with ambient noise level of 37 db. Dotted line indicates tests in same room with doors open, ambient noise level of om 45 to 57 db.



SPEECH RECEPTION TESTS			
Ambient Noise	#9	PB-50	SAI
37 DB	30	92%	57
Between 45-57 DB	32	74%	47

SPEECH RECEPTION TESTS			
Ambient Noise	#9	PB-50	SAI
37 DB	30	92%	57
Between 45-57 DB	32	74%	47

Chart 8. Case 3. R. C. Effects of ambient noise in testing a patient with otosclerosis (conductive deafness). Solid line indicates tests in room with ambient noise level of 37 db. Dotted line indicates tests in same room with doors open, ambient noise level of from 45 to 57 db.

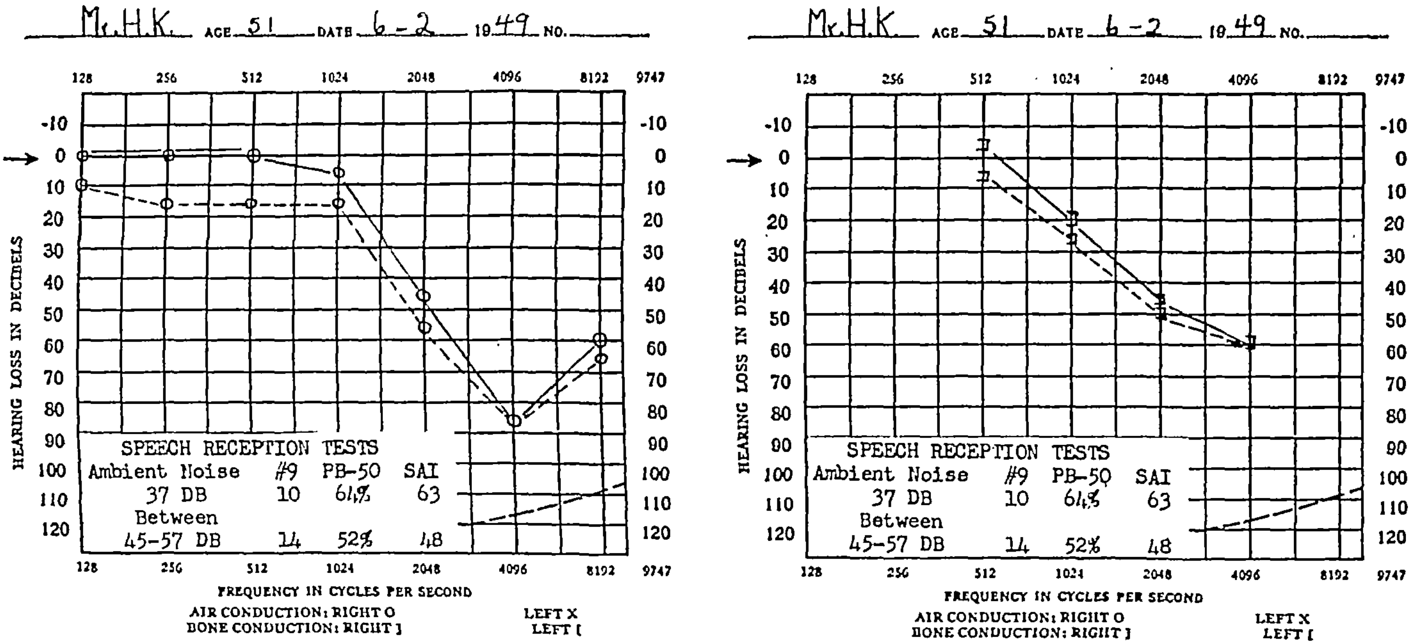


Chart 9. Case 4. H. K. Effects of ambient noise in testing a patient with nerve deafness (perceptive deafness). Solid line indicates tests in a room with ambient noise level of 37 db. Dotted line indicates tests in same room with doors open, ambient noise level of from 45 to 57 db.

the external auditory canal by an average of 20 db. below the ambient noise level of the room, providing tests are made in a room with an ambient noise level in the neighborhood of 1,000 cps. or above. A similar lowering may be produced by the use of one headphone and a dummy headphone, or a rubber plaster mixing bowl, clamped tightly over the ear not being tested.

For many years, attempts have been made to standardize bone conduction tests, but this has been practically impossible. In the first place, little is known about the transmission of sound through skin and bone; moreover, there is a wide variation in the structure of human heads. It has also been pointed out by physicists that sound waves are not easily transmitted from one medium to another of different acoustic resistance.[14]

The investigation with tuning forks, which has been described in this paper, shows that tests with tuning forks, due to the differences in their size and physical properties, have very little, if any, value; furthermore, the volume of sound produced varies with the method of activation and the position in which the tuning fork is held. In spite of making a great effort to activate the tuning forks with the same force, a variation of from 5 to 10 db. in the intensity of sound produced was noted in the investigation. In the opinion of the writer, tuning fork tests are of value only for gross comparisons, and their usefulness will be in direct proportion to the experience of the tester.

In the use of the electrically activated bone conduction receiver, some discrepancies are encountered; however, when the bone conduction receiver is properly standardized, reasonably dependable readings may be obtained for purposes of comparison.

Properly made recordings, when reproduced over a high fidelity system, afford a simple but reliable method of testing speech reception. All physical factors can be measured objectively. It is the nearest approach to objective evaluation of hearing loss that we have at the present time. It is, of course, impossible to measure objectively the purely nervous factor of hearing, which is subject to all the variables found in humans, and is obviously subjective in value. The cooperation of the patient is a factor that cannot be controlled. For this reason, the evaluation of hearing loss as determined with the use of recordings over a playback system will be in direct proportion to the cooperation of the patient, as well as to the reliability of the system and the experience of the tester, the latter two factors being controllable.

The monitored live voice has not been used in the tests. It is felt that the physical equipment required for its use is too elaborate. The same reliable evaluation of hearing loss can be obtained by using recordings, and the latter also have the advantage of permitting repeated tests at the same level without any question of human variables.

The Social Adequacy Index (SAI) for Hearing, which Hallowell Davis[3] prepared in his meticulous manner, and presented in 1948, fulfilled the long standing need for proper quantitative evaluation of hearing loss for speech. There had been need of such quantitative evaluation of hearing loss, not only for medicolegal purposes, but more particularly to determine the value of hearing aids, auditory training, and successful fenestration operations. The SAI for Hearing establishes the relationship between a patient's hearing loss for speech and the level of his plateau. For the first time, it is possible to give a patient an understandable prognosis. In the opinion of the writer, the SAI for Hearing will, in the future, be as valuable with relation to speech reception tests as the audiogram is with relation to pure tones tests.

CONCLUSIONS.

Hearing loss for speech and pure tones may be properly evaluated by using the following minimum physical equipment and tests:

1. *Physical Equipment:*

A sound treated room with an ambient noise level of not more than 45 db. when measured with the weighting curve C (equal response over entire range).

A modern audiometer equipped either with two headphones encased in firm rubber, or with one headphone and a dummy headphone, or a rubber plaster mixing bowl, over the ear not being tested.

A reliable playback system equipped with a trimmer and a zero level meter for the reproduction of recordings.

All equipment including recordings must be standardized under the conditions to be used in the tests.

The above equipment requires a financial outlay of less than $1,000.

2. *Essential Tests:*

Air conduction for pure tones from 128 cps. to 8,192 cps., the two extremes being of questionable value.

Bone conduction for 512, 1,024, 2,048, 4,096 cps., which furnishes a clue and helps to round out the overall picture.

Speech reception tests using:

Spondee words (Auditory Test 9) for the determination of threshold.

PB 50-word lists given at 25 db. above threshold for diagnostic purposes, and at the maximum intensity tolerated by the patient for prognostic purposes.

By means of the Social Adequacy Index (SAI) for Hearing, it is possible to quantitatively evaluate the hearing loss for speech.

In the final analysis, hearing tests to have any value must be meticuously performed under proper conditions. Such tests, when combined with the complete history of the case and physical examination, present a reliable basis for hearing evaluation.

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TUMORS OF THE TRACHEA.[*][†]

PAUL H. HOLINGER, M.D.; FRANK J. NOVAK, M.D.,
and KENNETH C. JOHNSTON, M.D.,
Chicago, Ill.

Tumors of the trachea are relatively rare. According to various authorities they range in frequency from one to three hundred, to one to eight hundred as compared to tumors of the larynx. They are not usually recognized until they are far advanced, because no localizing symptoms develop until they become large enough to produce severe respiratory obstruction.[28]

Twenty-eight cases of tracheal tumors seen at The Research and Educational Hospitals, St. Luke's Hospital and The Children's Memorial Hospital during the past 14 years are reviewed. They may be classified as nonspecific and specific inflammatory tumors, and benign and malignant neoplasms. Eighteen of the 28 tumors were primary in the trachea. The remaining 10 tumors invaded the trachea from adjacent structures to give signs, symptoms and endoscopic findings of primary tracheal tumors.

Summaries of the histories, findings and therapy are given in the accompanying tables. Table I lists the data of the patients found to have inflammatory tumors in the trachea. Five of the tumors are placed in this category; two are examples of nonspecific post-tracheotomy granulomas that caused respiratory obstruction which prevented extubation or required endoscopic removal after extubation. These two

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[*] Presented at the Fifty-fourth Annual Meeting, American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 27, 1950.

[†] From the Department of Broncho-Esophagology, the Children's Memorial Hospital, and the Divisions of Broncho-Esophagology, the Department of Otolaryngology, the University of Illinois College of Medicine, and St. Luke's Hospital, Chicago, Ill.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 21, 1950.

cases are representative of numerous similar cases in which the presence of granulomas around the tracheotomy tube at the point of the tracheostomy caused definite delay in extubation following tracheotomy. In one of the two cases exuberant granulation tissue was found to prevent extubation, the tracheotomy tube still being *in situ*. In the second case, a three-year-old boy, wheezing and dyspnea recurred 10 days after the tracheotomy tube had been removed. Lateral X-rays of the neck demonstrated a round shadow in the trachea at the site of the previous tracheostomy (see Fig. 1). Passage of the bronchoscope to this portion of the trachea demonstrated a granuloma almost completely obstructing the trachea. The tumor was dislodged with the tip of the bronchoscope and a normal airway was re-established. A similar coring technique was used in removing the granuloma from the trachea of the other patient, the tracheotomy tube being removed as the bronchoscope approached the tracheostomy.

These and other similar nonspecific inflammatory granulomas are of considerable clinical importance.[24] They are seen as tumors around foreign bodies of long standing within the trachea or bronchus, and as masses obstructing a bronchus leading to a segment of lung involved in a lung abscess. Their significance is that they produce both a respiratory obstruction and an obstruction to adequate drainage.

Three of the inflammatory tumors are the result of specific inflammatory disease. One of these tumors was a large tuberculoma that together with tuberculous ulceration and exudate eventually caused the death of the patient from tracheal obstruction. A second specific inflammatory tumor was due to torula. This tumor obstructed the trachea and right bronchus of a six-year-old boy. It was removed endoscopically. The following is the case report of this patient.

D. T. (Case 4, Table I), six years of age, male, was admitted to The Research and Educational Hospitals on Oct. 35, 1946. His symptoms began with wheezing in January, 1944, and he received treatment for asthma until it was discovered that breath sounds were absent in the right chest. Dyspnea gradually increased and a tracheotomy was done elsewhere in April, 1944. No relief of the dyspnea followed; therefore, the tube was removed a short time later. He continued to have recurrence of acute obstructive symptoms, but these moderated with penicillin

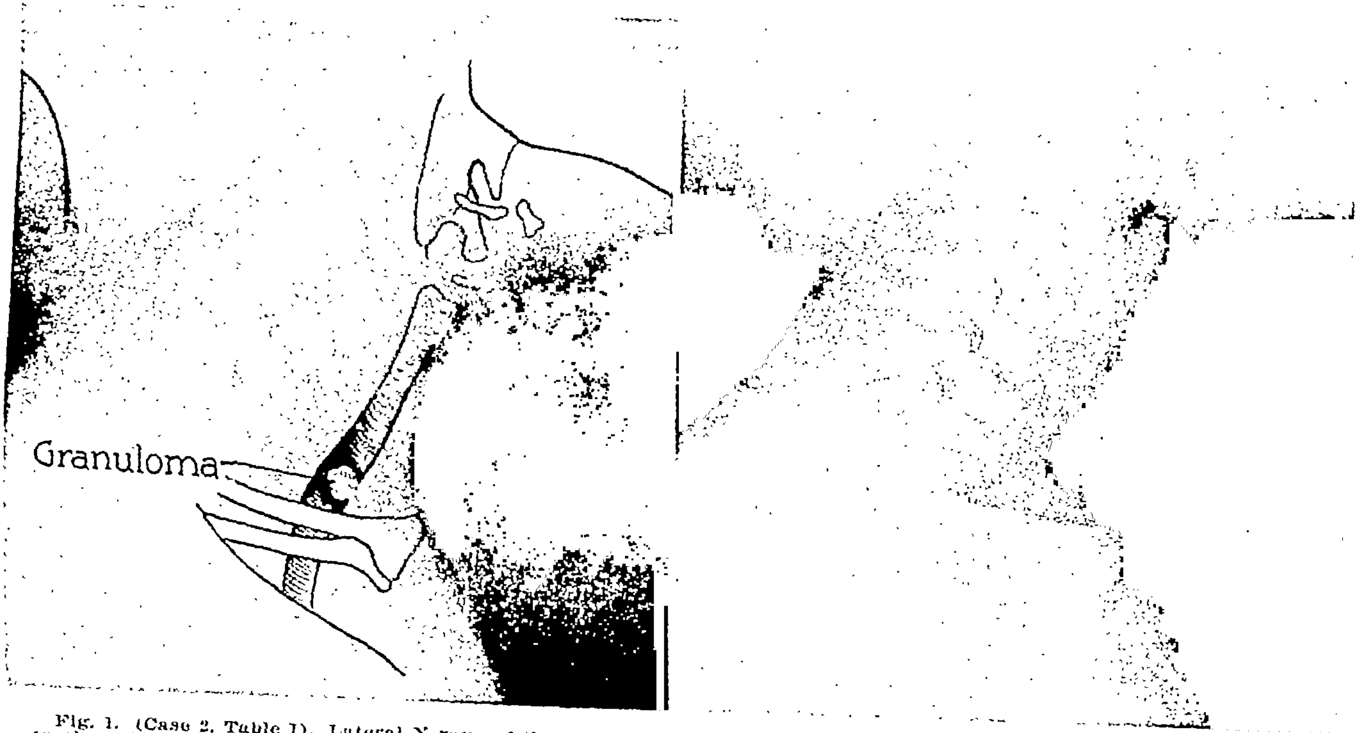

Fig. 1. (Case 2, Table I). Lateral X-rays of the neck demonstrating a nonspecific post-tracheotomy granuloma in the trachea at the site of the tracheostomy.

TABLE 1.

No.	Name and Sex	Age at Onset	Age on Admission	Symptoms and Laboratory	X-ray	Endoscopy	Therapy	Result	Diagnosis
1.	A.L. male	3 mos.	8 mos.	Tracheal obstruction; tracheotomy	Obliteration of shadow of airway proximal to tracheotomy tube.	Granuloma, anterior tracheal wall at tracheotomy.	Endoscopic removal.	Extubation following removal of granuloma; cured.	Post-tracheotomy granuloma
2.	J.B. male	3 yrs.	3 yrs.	Tracheal obstruction 10 days after extubation.	Granuloma at site of previous tracheotomy.	Granuloma at site of tracheotomy.	Endoscopic removal.	Cured.	Post-tracheotomy granuloma
3.	A.K. female	25 yrs.	26 yrs.	Wheeze, dyspnea; sputum positive for tuberculosis.	Bilateral pulmonary tuberculosis	Tracheal granuloma and exudate obstructing tracheal lumen.	Endoscopic removal. Biopsy: Tuberculoma.	Died.	Tuberculous granuloma of trachea
4.	D.T. male	4 yrs.	6 yrs.	[illegible], dyspnea, atelectasis, right lung. Torula grown from tissue removed from trachea.	Atelectasis, right lung, planograms demonstrate tracheal tumor	Mass above carina.	Endoscopic removal. Biopsy: Torula	Improved, trachea clear, bronchiectasis, rt. lung.	Torula of trachea
5.	T.L.L. male	36 yrs.	46 yrs.	Wheezing and hoarseness, tightness in throat.	Dense nodular infiltration of tracheal cartilages	Smooth, hard, nodular indentations into tracheal lumen.	None	Unchanged	Nodular calcification of tracheal cartilages

treatment. On Sept. 1, 1945, he was re-examined and found to have a complete atelectasis of the right lung; a bronchoscopic examination was made and tissue obtained from the trachea which established the diagnosis of Cryptococcus hominis (torula). At the time of admission to The Research and Educational Hospitals, approximately two months later, he had a persistent productive cough and was dyspneic and cyanotic. Physical findings and X-rays demonstrated an almost complete atelectasis of the right lung, with the apex beat of the heart to the right of the sternum, and an emphysema of the left chest. Laboratory studies showed hemoglobin 12.6, red blood cells 4,300,000 white blood cells 15,000, with an essentially normal differential count. The Kahn test was negative; blood chemistry studies, urinalysis and the electrocardiogram were normal. Skin and sputum studies, including guinea pig inoculation, were negative for tuberculosis. Histoplasma and coccidioidin skin tests were negative. Planographic studies demonstrated a tumor obstructing the distal trachea and entrance of the right main bronchus (see Fig. 2). There was no X-ray evidence of metastases to the spine or ribs. A bronchoscopic examination was made with oxygen flowing through the bronchoscope, and a large soft mass seen obstructing the tracheal lumen above the bifurcation. Sufficient tissue was removed to improve the airway and the procedure then discontinued. Histologic examination and tissue cultures confirmed the diagnosis of torula. A bronchogram three months later, after repeated bronchoscopic removal of obstructing tissue had established a normal lumen, showed a bronchiectasis of the right lung; however, after consultation with the department of thoracic surgery, a pneumonectomy was not advised because of the previous evidence of involvement of the trachea and right bronchus at the point at which the resection would have had to be done. This boy has remained symptom-free. During the month of December, 1945, this patient's father was also a patient in The Research and Educational Hospitals and succumbed to a brain abscess which followed pneumonia. No evidence of torulosis could be demonstrated on anti- or postmortem.

The fifth patient in the group of inflammatory tumors (see Case 5, Table I) had extensive calcification of the tracheal cartilages with nodular masses protruding into the lumen of the trachea (see Fig. 3). These were coverd by mucosa and presented the picture of tracheopathia osteoplastica. Careful medical and X-ray surveys showed no abnormal calcium metabolism and no unusual calcium deposits elsewhere.

Of the neoplastic tumors of the trachea reported in this series, five were benign and nine malignant. The benign tumors include one fibroma and one papilloma of the trachea, two amyloid tumors and one adenoma. The following three case histories of a fibroma, an amyloid tumor and an adenoma are representative of patients in this group:

W. L. (Case 7, Table II), 33-year-old white female, entered St. Luke's Hospital on Oct. 9, 1947. She gave a history of progressive trouble in breathing of one year's duration. For the previous two months the

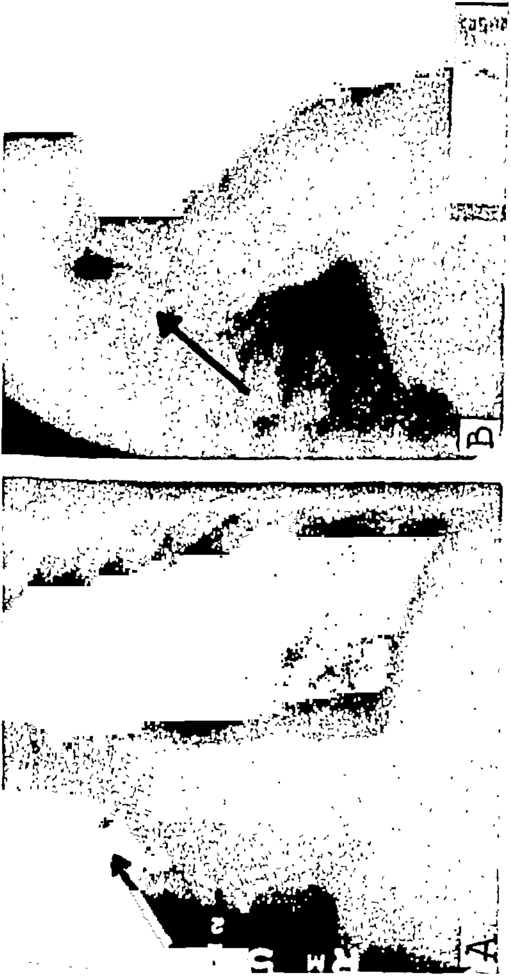

Fig. 2. (Case 4, Table 1). Planograms showing tumor due to torula obstructing the trachea and entrance to the right main bronchus.

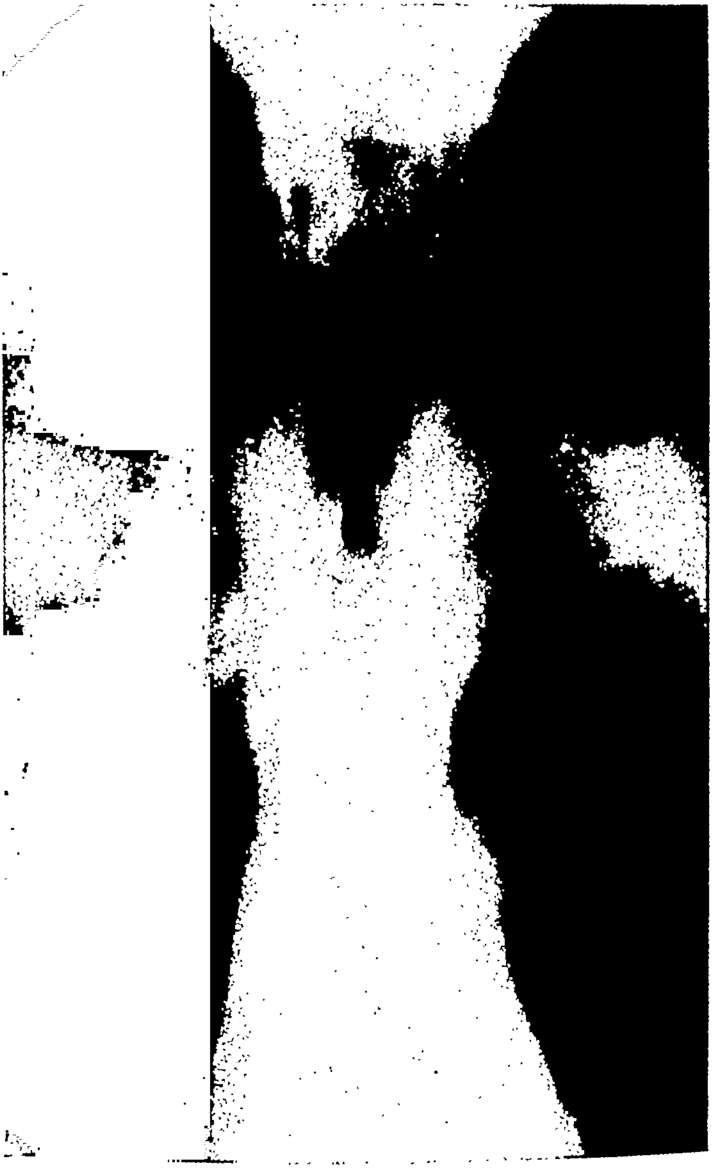

Fig. 3. (Case 5, Table I) Planogram showing nodular obstructing calcification of the tracheal cartilages.

patient had been unable to lie down to sleep. The dyspnea was primarily inspiratory. She was taken to the operating room on admission, and an emergency tracheotomy was performed, using a Mosher lifesaver to

TABLE 2.

No.	Name and Sex	Age at Onset	Age on Admission	Symptoms and Laboratory	X-ray	Endoscopy	Therapy	Result	Diagnosis
6.	B.K. male	57 yrs.	59 yrs.	Hoarseness, hemoptysis, wheezing respiration leukemia	Chest film negative	Mass occluding trachea, 17 cm. from upper teeth. Biopsy: Papilloma	Endoscopic forceps removal	No recurrence of tracheal tumor 4 yrs. later. Died of leukemia.	Papilloma of trachea; chronic lymphatic leukemia
7.	F.L. female	32 yrs.	33 yrs.	Cough; dyspnea	Mass on post-tracheal wall below cricoid cartilage	Pedunculated, round mass attached to post-tracheal wall. Biopsy: Fibroma	Endoscopic forceps removal. Tracheotomy	Cured. Tracheotomy tube removed.	Fibroma of Trachea
[illegible]	L.D. male	72 yrs.	74 yrs.	Asthmatic wheeze; acute respiratory obstruction	None pre-operatively because of acute obstruction. Chest film negative.	Soft mass in mid-trachea "cored" with bronchoscope. Biopsy: Amyloid Tumor	Endoscopic "coring" with bronchoscope to establish lumen. Later forceps removal.	Improved. Residual amyloid tissue.	Amyloid Tumor of Trachea
[illegible]	L.F. female	34 yrs.	40 yrs.	Wheeze, dyspnea, cough. Previous history of positive sputum. Sputum negative on admission	Homogeneous density, rt. upper lobe	Narrowing of tracheal and bronchial walls by soft friable tissue. Occlusion, rt. upper lobe bronchus.	Endoscopic removal by coring and forceps. X-ray therapy	Improved. Residual amyloid tissue.	Amyloid disease of trachea and bronchi.
10.	J.T. female	37 yrs.	43 yrs.	Wheeze, cough, dyspnea, orthopnea, "Status asthmaticus"	Chest X-ray negative. Planograms showed round tumor in mid-trachea	Smooth, pedunculated round mass occluding mid-trachea. Biopsy: Adenoma	Endoscopic forceps removal. Electro-resection.	Improved. Residual adenoma.	Adenoma of Trachea

establish the airway during the procedure. A No. 5 tracheotomy tube was inserted in the midline of the trachea. Subsequent examination showed a tumor attached to the right posterolateral wall of the trachea at the level of the second ring. This was removed with cup forceps at its pedicle. One week later a residual 3 mm. nodule was removed from the site of the tumor and the base electrocoagulated. The pathologist reported the tumor to be a fibroma with lipid deposits and reactive fibroplastic tissue, and suggested the possibility that the tumor might be a metastasis from the kidney. Pyelograms and urinalyses revealed no disease of the genitourinary tract, and complete blood studies for lipid metabolism revealed no abnormality. The blood count and Kahn test were normal. The patient has been well following the removal of the tumor.

L. D. (Case 8, Table II), a 74-year-old white male, entered St. Luke's Hospital, Feb. 13, 1948. He gave the history of having experienced difficulty in breathing for two years. At the onset the dyspnea occurred with colds and was relieved by adrenalin. During the next few months the attacks became more severe, though not more frequent, and adrenalin became ineffective. In December, 1947, a very severe attack occurred, followed by complete relief for two months. In the week previous to admission the patient had had three severe attacks of dyspnea and cyanosis. On arrival at St. Luke's Hospital as an emergency, the patient became unconscious and was taken immediately to the operating room. A bronchoscope was passed through the larynx without anesthesia; 5 cm. below the cords the trachea was found to be almost completely obstructed by soft, pale tissue. The bronchoscope was advanced through the area into the right bronchus, "coring" the mass to create a lumen; the core was removed from the bronchoscope and the airway thus re-established. With oxygen and artificial respiration normal breathing was gradually resumed. The histologic report of the tissue obtained was "amyloid deposits in tracheal tissue." The patient was then given Congo red and subsequent tissue removed bronchoscopically was supravitally stained red with the dye. This tissue also gave the iodine test for amyloid. Five days later bronchoscopy was repeated, and large pedunculated pieces of tumor were removed, and the airway enlarged. A third bronchoscopy was performed one week later. The entire circumference of the trachea was involved as far as the coryna.

Urinalysis revealed an albuminuria and occasional hyaline casts. The blood count was normal. Blood chemistry revealed 60 per cent Congo red in the blood; this indicated 40 per cent retention, an abnormal finding. Bronchoscopy eight months after discharge from the first admission revealed a roughened trachea with good airway. No other site of amyloid infiltration has been found in this patient.

S. S. (Case 10, Table II), a 43-year-old white woman, entered St. Luke's Hospital, Jan. 24, 1949. She gave a history of having had difficulty in breathing, with asthmatic wheezing for the past six years. Breathing had become increasingly difficult during the past year. Three series of allergy tests had been made because of persistent asthma. There was a slight nonproductive cough. A bronchoscopic examination had been made elsewhere, at which time a tracheal tumor had been found almost completely obstructing the trachea, and the patient was referred for removal of the tumor. X-ray studies prior to bronchoscopy by planographic technique in the anteroposterior and lateral views showed a round tumor 1 cm. in diameter attached to the posterior wall of the trachea in its midportion (See Fig. 4. Bronchoscopic examination showed the tumor to be round, slightly nodular and covered with smooth mucosa with larg

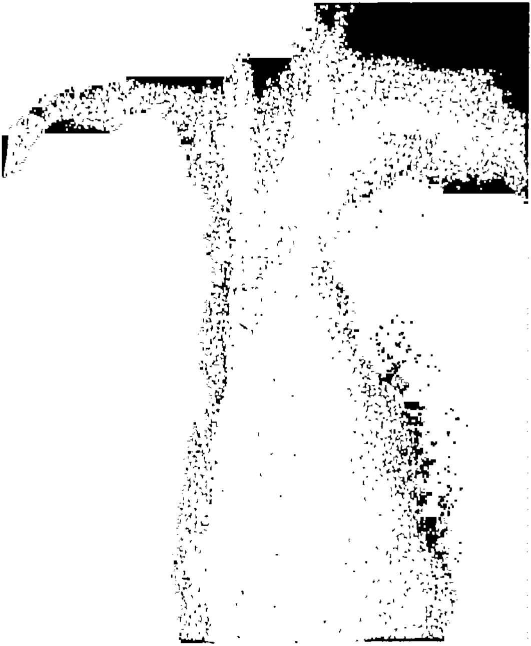

Fig. 4. (Case 10, Table II). Planogram in A-P projection at 6 cm. level showing round tumor obstructing the midportion of the trachea. Histologic study showed the tumor to be an adenoma.

vessels on its surface. Except for a small slit on the anterior and left tracheal wall the tumor obstructed the entire lumen of the trachea. The 12 mm. photographic bronchoscope was passed directly over the tumor, removing it nearly *in toto*. An attempt to coagulate the base was interrupted by bleeding. The examination was continued three days later, further tissue removed and the base of the tumor coagulated. The tumor proved to be an adenoma of the trachea. Laboratory data were essentially normal. The patient is asymptomatic at this time, but tracheoscopy has demonstrated residual adenomatous tissue on the posterior tracheal wall. Further tissue has been removed with the resectascope,[33] using the cutting loop and then coagulating the base of the lesion with the electrocoagulating current (see Fig. 5).

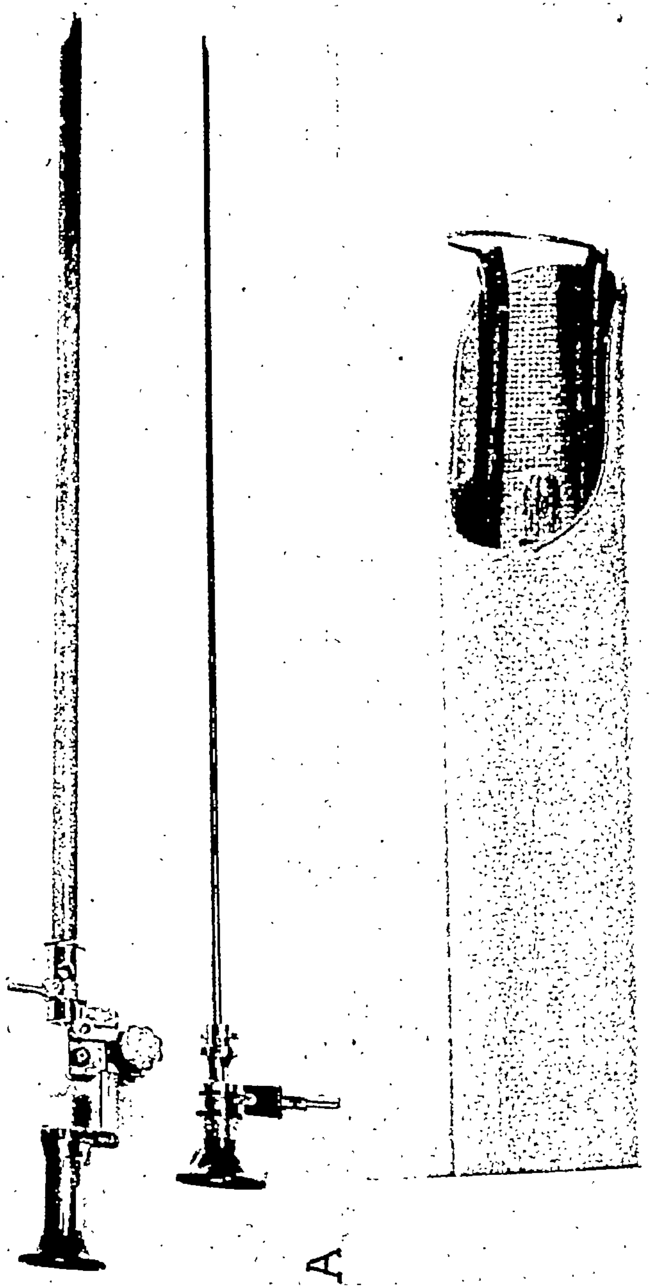

Fig. 5. (A) Resectoscope similar to cystoscopic resectoscope used with cutting loop or coagulating tip in cases of tracheal tumors. (B) Close-up of tip of scope and cutting loop.

Of the benign tumors of the trachea reported in the literature, the fibroma appears to be the most frequent,[3,10,28,31,42] and the papilloma[6,13,30] next in order of incidence. In patients who have papilloma of the trachea the primary source is almost always found in the larynx, the tracheal growths representing either extension of the growths downward or isolated transplants. Primary papilloma of the trachea without laryngeal involvement is rare.[6,24] Amyloid tumors,[45] tracheopathia osteoplastica,[10,38] and adenomas which are difficult to classify,[44,48] complete the list of benign neoplastic tumors of the trachea encountered, although lipomas, angiomas,[23] and chondromas are occasionally seen.[39]

The nodular tumors of rhinoscleroma should be included in this discussion.[37,16] Although these have not been seen in the cases herein reported, they are being seen with increasing frequency in this country.[34]

The malignant tumors constitute the largest group of primary tracheal tumors both from the standpoint of a review of the literature and from the cases herein reported.[2,4,11,13,14,16,17,20,33,40,47] Five of the nine primary malignant tumors in this series were squamous cell carcinoma, one was an adenocarcinoma, and one a cylindroma. Two cases of myosarcoma of the trachea, previously reported,[22] are included for statistical purposes. The history of a case representative of this group is as follows:

W. R. (Case 11, Table III), 45-year-old white male, entered the Research and Educational Hospitals on Sept. 25, 1945. He stated that he first noted difficulty in breathing and exercising during the previous winter. A severe cough was associated with wheezing and occasional hemoptysis. In April, 1945, a biopsy was taken elsewhere from the posterior wall of the trachea below the larynx, establishing the diagnosis of carcinoma of the trachea. Thirty-four X-ray treatments (4,250 r.) were given and in March, 1945, a tracheotomy was done for increasing dyspnea. The lesion progressed and 18 additional X-ray treatments (3,600 r.) were given. A direct laryngoscopy and tracheoscopy on Oct. 2, 1945, revealed tumor tissue on the posterior wall of the trachea, partly obstructing the airway. Biopsy showed persistent anaplastic squamous cell carcinoma. Serology and red and white blood cell counts were normal. Since no response had been obtained with X-ray, the patient was surgically explored on Oct. 16, 1945, in an attempt to remove the larynx, upper trachea and esophagus. Mediastinal glands and extension to the esophagus were discovered and the operation discontinued. The patient had progressive difficulty as the carcinoma extended further into the esophagus, and when he could no longer maintain adequate nutrition orally a Witzel gastrostomy was done. This was performed on March 12, 1946, over a year after the onset of his symptoms. He expired on April 15, 1946.

The malignant tumor encountered most frequently in this series is squamous cell carcinoma (see Fig. 6). In other reports of series of cases, adenocarcinoma appears to be the most frequent malignant tumor. Only one of these has been encountered in our series. While the life expectancy of this group generally has been relatively poor, due to early invasion of adjacent vital structures and to metastases, occasional patients whose tumor is detected early will respond to local treatment and may be considered eligible for tracheal resection by some of the newer techniques. The following case report is of interest because of the duration of life after establishment of the diagnosis:

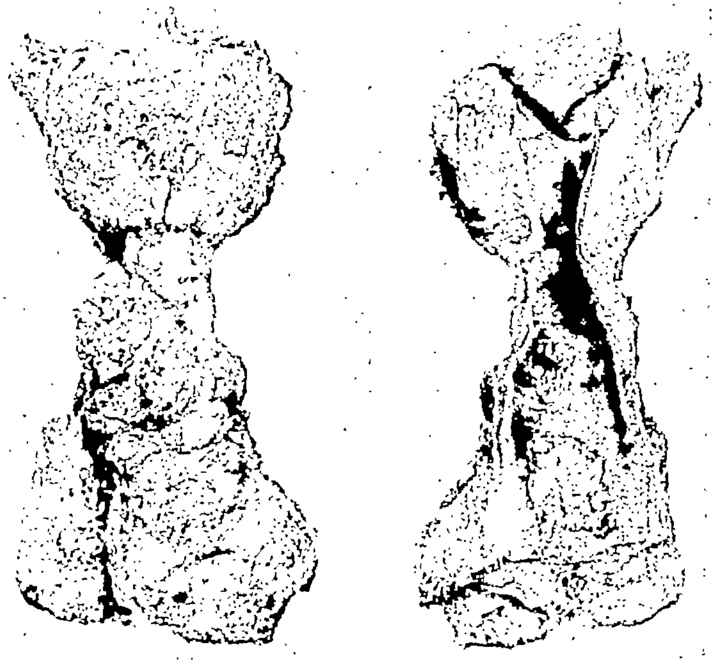

Fig. 6. (Case 14, Table III). Squamous cell carcinoma of the trachea. The collar-button character of the tumor is shown, the small intratracheal mass giving little evidence of the extratracheal extent of the tumor.

C. G. (Case 15, Table III), 52-year-old white male, entered St. Luke's Hospital for the first time July 2, 1943, complaining of an incessant, uncontrollable, nonproductive cough of one year's duration, an unexplained fever of six weeks' duration, and some weight loss. Complete blood counts, urinalysis and Kahn test were normal. A bronchoscopic examination was made July 3, 1943, which revealed a lobulated, somewhat pedunculated mass attached to the anterior tracheal wall, 2 cm. above the bifurcation and descending to the right main bronchus. Biopsy showed the tumor to be a squamous cell carcinoma of the trachea, and resection and electrocoagulation of all visible tissue were done. *On the second admission, Feb. 8, 1945, bronchoscopy showed slight* roughening of the trachea but no evidence of tumor tissue. On Oct. 23, 1946, a soft, friable tumor was again present on the anterior wall of the trachea and the biopsy was positive. The area of involvement was again extensively fulgurated after forceps removal of all pedunculated tissue. One month later reinspection showed the trachea to be smooth and the airway normal, but on March 15, 1947, a 1 cm. area on the anterior tracheal wall showed evidence of recurrence; this was fulgurated. By Dec. 10, 1947, partial obstruction of the trachea developed and this was reduced by bronchoscopic electrocoagulation; again on Jan. 23, 1948, the anterior wall was coagulated 2 cm. above the bifurcation. Respiratory difficulty occurred three and one-half months later and on May 12, 1948, the tumor was found to occlude one-half of the lumen of the trachea with compression of the right main bronchus. The airway was cleared by repeated bronchoscopic procedures. On May 28, 1949, one year later, bronchoscopy was done because of severe dyspnea. The patient was given nitrogen mustard intravenously, 6 mgm., four times in eight days, on this *admission; repeated bronchoscopic procedures with removal of* large amounts of tumor tissue were done to maintain the airway. Electrocoagulation and cup forceps were used to open the left main bronchus although the right side was still occluded. By June 22, 1949, a good view of the coryna and both main bronchi was obtained after removal of tissue from the anterior tracheal wall; eight 1.5 mc. radon seeds were injected into the tumor site bronchoscopically. Acute respiratory obstruction recurred frequently which required emergency aspiration and removal of tissue. Tracheotomy was considered, but because of the bilateral extent into the bronchi was not thought feasible. Guanazola 500 mgm. daily and ACTH 175 mgm. were both given in July, 1949, preceded by a series of external X-ray treatments, three ports, a total of 3,185 for a calculated tumor dose of 4,190 r. On Nov. 19, 1949, a paralysis of the left vocal cord developed and it was found that the tumor extended up the trachea more than had been noted previously. The coryna was broad and eroded by the tumor. On Dec. 31, 1949, the tumor had advanced to a point 23 cm. from the upper teeth. Nine 1.5 mc. radon seeds were inserted into the tumor after resection of tissue with the bronchoscopic resectascope. Tissue removed from the right vocal cord on Dec. 28, 1949, showed squamous cell carcinoma of the larynx, apparently a separate carcinoma from that in the tracheobronchial tree. Recently metastatic glands in the right supraclavicular area have caused a partial paralysis of the right recurrent laryngeal nerve. At present there appears to be a *temporary arrest of the growth although severe obstruction due* to tracheal destruction is present.

Cylindroma of the trachea is reported frequently in the literature.[4,19,27,33,36] No primary cylindroma of the trachea has been encountered in this series, although one patient previously reported[18] (Case 17, Table III) died of obstruction of the trachea by a cylindroma of the right bronchus that advanced upward into the trachea. This patient had, in addition, multiple metastases to both lungs, and had been under observation for seven years; the airway had been repeat-

TABLE 3.

No.	Case and Sex	Age at Onset	Age on Admission	Symptoms and Laboratory	X-ray	Endoscopy	Therapy	Result	Diagnosis
11.	W.R. male	45 yrs.	45 yrs.	Dyspnea, hoarseness, weight loss, cough and hemoptysis	Negative chest film	Mass occluding trachea from subglottic space to 6th tracheal ring. Biopsy positive.	X-ray. Attempted surgery. Tracheotomy.	Died 18 mos.	Squamous cell carcinoma of the trachea
12.	F.F. male	57 yrs.	57 yrs.	Dyspnea, cough and hemoptysis.	Chest x-ray negative	Mass in distal half of trachea partially occluding lumen	X-ray therapy	Died 7 wks.	Squamous cell carcinoma of the trachea
13.	E.P. male	49 yrs.	49 yrs.	Cough, hemoptysis, "Flu"	Chest X-ray negative.	Tumor subglottically on tracheal wall	X-ray therapy	Died 4 mos.	Squamous cell carcinoma of the trachea
14.	P.M. female	45 yrs.	45 yrs.	Cough, hemoptysis, dyspnea, chest pain.	Density, right lower lobe	Fibrous, irregular tumor 20 cm. from upper gums.	Tracheotomy; X-ray therapy	Died 5 mos.	Squamous cell carcinoma of the trachea
15.	C.O. male	52 yrs.	52 yrs.	Intractable cough; no hemoptysis	Chest film negative.	Friable, granular mass on anterior tracheal wall 2 cm. proximal to carina	Endoscopic resection; X-ray therapy; radon seed implantation; ACTH, etc.	Symptom-free for 5 years. Gradual progression of disease.	Squamous cell carcinoma of the trachea
16.	G.P. male	48 yrs.	49 yrs.	Cough, dyspnea, hemoptysis	Mediastinal mass in right hilus	Mass on right lateral wall above carina	X-ray therapy	Died 10 mos.	Adenocarcinoma of trachea
17.*	L.K. female	47 yrs.	53 yrs.	Cough, dyspnea, hemoptysis	Atelectasis, right upper lobe	Mass in trachea and right bronchus	Endoscopic removal; X-ray therapy.	Died 5½ yrs.	Cylindroma of the trachea and right bronchus.
18.**	C.L. female	3½ yrs.	5 yrs.	Asthmatic wheezing, dyspnea.	Chest negative. Lateral X-ray of neck: round mass on anterior tracheal wall.	Firm, round mass on anterior tracheal wall 4 cm. below larynx.	Surgical removal of trachea; plastic reconstruction	Living and well 4 yrs.	Myosarcoma of the trachea
19.**	F.C. female	5 yrs.	6 yrs.	Wheezing, acute dyspnea	Bilateral emphysema.	Friable mass in distal trachea and both bronchi	X-ray therapy (discontinued because of severe leucopenia)	Died 6 wks.	Myosarcoma of the trachea

edly re-established by bronchoscopic removal of obstructing tissue. Sarcoma and fibrosarcoma of the trachea are relatively rare,[1,41,50] and review of one case in this series originally thought to be a fibrosarcoma has led to its reclassification as a myosarcoma.[33]

Malignant tumors of adjacent structures which have invaded the trachea are included in this resumé of cases of tracheal tumors because of their similarity to primary tracheal tumors from the standpoint of history, X-ray and endoscopic findings. Ten carcinomas of adjacent structures were found invading the trachea to give primary symptoms and endoscopic findings of tracheal tumors. Eight of these were thyroid carcinomas, and two were esophageal carcinomas that had extensively invaded the trachea to cause respiratory obstruction. The following case reports are representative of this group:

J. A. (Case 23, Table IV), a 55-year-old white man, entered The Research and Educational Hospitals on July 10, 1945. He had been referred to the hospital because of dysphonia and stated that he had been hoarse for two and one-half months and had lost 15 pounds in weight. Mirror examination of the larynx revealed a bilateral recurrent laryngeal nerve paralysis, and inspection of the trachea showed a friable mass nearly occluding the trachea below the larynx. Tissue removed at this time showed the tumor to be a carcinoma of the thyroid. Because of the severe dyspnea a tracheotomy was done on Sept. 17, 1945. From Sept. 20, 1945, to Oct. 12, 1945, the patient received 4,700 Roentgens to the thyroid region through three portals. He progressed rapidly downhill and died less than five months after the onset of his symptoms.

M. D., (Case 28, Table IV), 54-year-old white woman, was admitted to St. Luke's Hospital on Sept. 22, 1949. She had been well until February, 1949, when she developed asthmatic breathing and, on careful questioning, stated that she had had some dysphagia and gagging. The dysphagia continued until September, 1949, but her principal difficulty was an increase in the noisy breathing and shortness of breath. This was treated as bronchial asthma with no success. At the time of admission to St. Luke's Hospital, Sept. 22, 1949, the patient was in constant respiratory distress and a bronchoscopic examination was made immediately in an effort to open the airway. A large tumor was found occluding the trachea and appeared to be attached to the right posterolateral wall of the trachea, 4 cm. distal to the larynx. A tracheoscope was passed, and the main portion of the tumor removed. Immediate relief from dyspnea resulted. It was believed that this was either a primary tracheal tumor or an erosion from the esophagus or thyroid. The biopsy showed the tumor to be a squamous cell carcinoma. Subsequent barium esophagrams and esophagoscopy revealed the primary lesion to be in the upper esophagus. Laboratory studies demonstrated a hypochromic anemia. Following the endoscopy the patient was given 1,170 r. to the right and left lateral neck ports. The subsequent course was one of progression of the disease which required a gastrostomy for feeding purposes. The patient died approximately three months after the diagnosis was established.

TABLE 4.

No.	Name and Sex	Age at Onset	Age on Admission	Symptoms and Laboratory	X-ray	Endoscopy	Therapy	Result	Diagnosis
20.	F.B. male	67 yrs.	69 yrs.	Hoarseness, dyspnea, dysphagia	Tracheal and esophageal compression above level of clavicles.	Friable mass 3 cm. below larynx. Biopsy: Adenocarcinoma of thyroid	Attempted thyroidectomy. Radon seed. Gastrostomy	Died 2 yrs.	Carcinoma of thyroid
21.	O.R. male	46 yrs.	55 yrs.	Hoarse; mass in neck; dysphagia; stridor	Anterior compression of cervical trachea.	Paralyzed right cord. Mass in trachea 4 cm. below larynx. Biopsy: Squamous cell carcinoma of thyroid	X-ray therapy. Gastrostomy	Died 4 mos.	Carcinoma of thyroid
22.	J.A. male	55 yrs.	55 yrs.	Hoarseness; weight loss of 15 pounds.	Mass compressing and invading cervical trachea.	Bilateral vocal cord paralysis. Mass partially occluding trachea at level of clavicles. Biopsy: Carcinoma of thyroid	Tracheotomy X-ray therapy	Died 5 mos.	Carcinoma of thyroid
23.	R.T. female	49 yrs.	65 yrs.	"Tumor in thyroid", wheeze, dyspnea	Compression and displacement of trachea to left with invasion of tracheal wall.	Friable mass in trachea below cricoid cartilage. Biopsy: Carcinoma of thyroid	Thyroidectomy; Tracheotomy; Radon seed implantation; X-ray therapy	Died 22 yrs. after original diagnosis of carcinoma of thyroid; 1 yr. after tracheal invasion	Carcinoma of thyroid
24.	J.B. female	73 yrs.	75 yrs.	Cough, wheeze, hemoptysis, weight loss	Deviation of trachea to left at level of clavicles.	Mass on right side of trachea, region of clavicles. Biopsy: Carcinoma of thyroid	Tracheotomy; X-ray therapy	Died 2 yrs.	Carcinoma of thyroid
25.	S.L. female	52 yrs.	53 yrs.	Progressive dyspnea; no hemoptysis	Marked distortion and narrowing of trachea with slit-like lumen far to left of midline	Mass in trachea below level of larynx; complete occlusion. Biopsy: Squamous cell carcinoma of thyroid	Tracheotomy; X-ray therapy	Died 1 yr.	Carcinoma of thyroid
26.	K.S. female	49 yrs.	52 yrs.	Dyspnea; mass in neck.	Posterior and left lateral displacement of cervical trachea.	Paralysis, rt. vocal cord; deviation of trachea to left. Papillary projection on rt. tracheal wall. Biopsy: Carcinoma of thyroid	Thyroidectomy; X-ray therapy; Radon seed implantation; Tracheotomy	Died 3 yrs.	Carcinoma of thyroid
27.	M.K. female	47 yrs.	51 yrs.	Hoarseness; mass in neck; dyspnea; cough; hemoptysis	Mass in trachea on lateral neck film	Paralysis, right cord. Friable bleeding mass at level of 2nd ring occluding trachea	Tracheotomy; X-ray therapy; Thyroidectomy	Died 4 yrs.	Carcinoma of thyroid
28.	L.D. female	54 yrs.	54 yrs.	Dyspnea; orthopnea; wheezing; "asthmatic attacks"; dysphagia	Irregular filling below cricoid & swelling of post-tracheal wall.	Friable mass in upper trachea. Esophagoscopy: Mass in esophagus. Biopsy: Squamous cell carcinoma of esophagus; tumor in trachea and esophagus same histology.	X-ray therapy; Gastrostomy	Died 7 mos.	Carcinoma of Cervical Esophagus

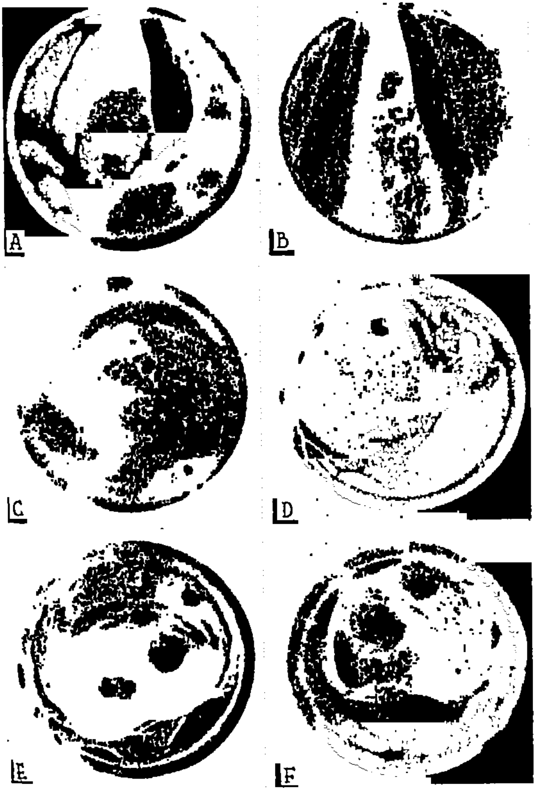

Fig. 7. Endoscopic photographs of various tracheal tumors. (A) (Case 7, Table II). Pedunculated fibroma attached to the posterior wall of the trachea at the level of the second tracheal ring. (B) (Case 11, Table III). Carcinoma of the cervical trachea extending upward into the subglottic space. (C) (Case 16, Table III). Adenocarcinoma of the distal trachea. (D) (E) (F) Carcinoma of the coryna with seeding of carcinoma along the walls of the trachea. (D) Level of the first ring; (E) midtrachea; (F) 1 cm. above coryna.

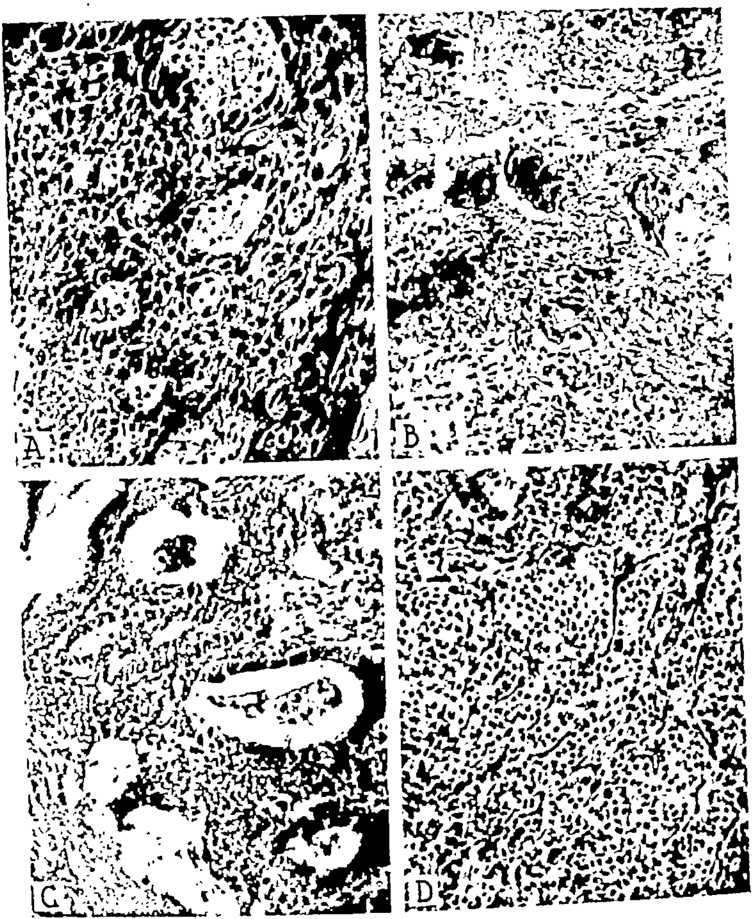

Fig. 8. Photomicrographs of four representative tracheal tumors. (A) (Case 11, Table III). Preliminary squamous cell carcinoma of the trachea. (B) (Case 4, Table I). Torula of the trachea. (C) (Case 16, Table III). Adenocarcinoma of the trachea. (D) (Case 10, Table II). Adenoma of the trachea.

DISCUSSION.

In most cases in this series the first symptom noted was a hacking, dry, constantly irritating cough that could not be relieved with cough sedatives or expectorants. The cough was frequently associated with hemoptysis which varied from slight blood streaking to gross hemorrhage, most pronounced in the malignancies. Stridor was another constant symptom which at first was quiet and positional and later became a loud, asthmatic expiratory wheezing which was bilateral and so clearly simulated asthma that most of the patients had had thorough and repeated allergy studies and treatment for asthma. Dyspnea and orthopnea were present in the late cases, and hoarseness occurred in the malignancies when vocal cord paralysis developed because of outward extension or mediastinal involvement. Dysphagia was occasionally noted.

The history of the symptoms noted above as well as the physical findings were exceedingly important in establishing the diagnosis in the patients in this series. The X-ray studies included inspiration and expiration chest films, lateral X-rays of the neck and trachea, planograms in the anteroposterior and lateral planes, and bronchographic studies. The final diagnosis was made by the bronchoscopic examination (see Fig. 7) and the removal of tissue for biopsy (see Fig. 8).

The first problem in treatment was usually the establishment of the airway. This frequently was an emergency procedure consisting of coring the tumor, removal of obstructive tissue by forceps or snare, and occasionally a tracheotomy, the tube being inserted below the tumor or through or beyond it by the use of a long tracheotomy tube. Endoscopic electroresection and coagulation were important endoscopic procedures in keeping the airway free of tissue.[13] Radon implantation and external irradiation were adjuncts in the treatment of the malignancies. Tracheal resection was accomplished in one case of myosarcoma of the trachea to give a five-year cure[24] and will undoubtedly assume a more important rôle in future cases if early diagnosis can be accomplished. The experimental work on intrathoracic tracheal resection gives promise of a new procedure available for selected cases of tracheal tumors.[9]

SUMMARY.

1. The clinical data of 28 cases of tracheal tumors are presented. Eighteen of the tumors were primary in the trachea, 10 invaded the trachea from adjacent structures to give signs, symptoms and endoscopic findings of

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EPIDERMOID CARCINOMA OF THE LARYNX OCCURRING IN TWO CHILDREN WITH PAPILLOMA OF THE LARYNX.

THEO. E. WALSH, M.D.,[†]
St. Louis, Mo.,
and
PARKER R. BEAMER, Ph.D., M.D.,[*]
Winston-Salem, N. C.

INTRODUCTION.

Papilloma of the larynx, the most common of all benign tumors in this organ, may be classified as either of two types on the basis of clinical observations and the histologic pattern of the lesion: *a.* a focal or diffuse hyperplastic thickening of epithelium with prominent hyperkeratosis, usually presenting a fairly regular and evenly contoured surface; *b.* a focal or diffuse, fungating hyperplastic thickening of epithelium, usually without a significant amount of keratosis, and presenting an irregularly contoured, rugose surface. The former, often called "hyperkeratotic papilloma," is usually encountered in adults and occasionally precedes the development of carcinoma of the larynx, particularly in men beyond the fourth decade of life. The second type, frequently called "juvenile papilloma," is usually found in infants and children, and seldom precedes the development of a malignant tumor of the larynx.

Owing to the apparent rarity of carcinoma developing at or in the site of papilloma of the larynx in a child, the following discussion of two cases seems to be deserving of consideration:

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[*] Departments of Pathology and Microbiology, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, N. C.

[†] Department of Otolaryngology, Washington University, School of Medicine and McMillan Hospital.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 21, 1950.

CLINICAL OBSERVATIONS.

Case 1: D. M., a white boy, 12 years old, was admitted to the St. Louis Children's Hospital for the first time, at the age of 16 months, with a history of hoarseness and diminution in voice beginning when the infant was 10 months old. During the six months prior to the first admission these symptoms had progressed and the child developed difficulty with respiration. Examination revealed a profuse growth of papilloma in the larynx. The tumor was removed through a Jackson laryngoscope, and the patient was discharged a few days later. Two months thereafter the child (age, 18 months) was admitted for the second time, and recurrent papilloma was removed by the Lynch suspension method. From this period, until the child was 39 months old, he was admitted to the hospital 11 times more for removal of rapidly recurring papilloma, the admissions varying between one and six months apart. During some of the admissions (between the ages of 30 and 35 months), in addition to surgical removals of tumor, the patient was treated by X-radiation to the larynx according to the following schedule:

Date	Age	Port	Kv.	Filter	Dist.	M.A.	Time	Units
6/29	30 mo.	50 sq. cm.	200	1.0 cu. lal.	50 cm.	15	18 min.	670 R.
8/ 7	32 mo.	65 sq. cm.	200	0.5 cu. lal.	50 cm.	15	9 min.	675 R.
8/ 8	32 mo.	65 sq. cm.	200	0.5 cu. lal.	50 cm.	15	9 min.	675 R.
10/19	34 mo.	65 sq. cm.	200	0.5 cu. lal.	50 cm.	15	9 min.	675 R.
11/12	35 mo.	65 sq. cm.	200	0.5 cu. lal.	50 cm.	15	9 min.	675 R.

Total X-radiation = 3270 Roentgen units.

During the series of admissions shown in the above schedule, tracheotomy was performed (age, 34 months) and the cannula was worn continuously for the remaining nine years of the child's life. The patient was brought to the hospital for his fourteenth admission when he was seven years old, and recurrent papilloma was removed from the larynx and trachea.

The boy was not under physician's care again until one of the present authors (T. E. W.) examined him for the first time, during the child's fifteenth admission (age, 11 years). At this time the tracheotomy wound was infected, and the patient had difficulty with swallowing and regurgitated fluids through the tracheotomy. The larynx was filled with papilloma, and tracheoscopy revealed a tracheoesophageal fistula. Biopsy of the tumor revealed interesting histologic findings discussed below. During this admission, and the sixteenth and seventeenth which followed four and nine months later, respectively, various methods of therapy were tried. Topical and parenteral estrogenic medication caused no apparent change in the tumor. The infection was treated with penicillin and sulfonamides. During the fifteenth admission gastrostomy was performed, and closure of the tracheoesophageal fistula was attempted by a plastic surgeon by turning a flap of skin between the trachea and esophagus. This procedure was accomplished from one side, and, owing to an unsatisfactory result, another skin-flap was brought in from the opposite side in a second repair. The patient was discharged shortly thereafter with slight improvement in his general condition.

The eighteenth and final admission, about one month later (age, 12 years), was occasioned by recurrent severe vomiting, loss of weight, and generally poorer condition of the boy. Technically, the second plastic repair was successful, but tumor had involved the area in and about the operative wound. This region was infected, also, and there were draining sinuses in the neck. Following blood transfusions and proper diet, there was temporary improvement in the patient's general condition. Sanguineous exudate and blood began to ooze from the sinuses, and, despite supportive therapy, the child died from a massive hemorrhage on the twenty-second hospital day.

A large series of biopsies, taken during the 18 hospital admissions, revealed interesting histologic findings which are presented below with a brief summary of the autopsy observations.

Case 2: V. S., a white girl, was admitted to the St. Louis Children's Hospital for the first time when she was three years old. The parents stated that the child suffered "frequent attacks of croup" since infancy. Six months prior to admission she lost her voice and was treated by a physician who "removed growths from the larynx"; this procedure was repeated about three months later. Upon admission to the hospital the larynx was full of papilloma of the "juvenile type," the airway was markedly restricted, and tracheotomy was performed to give the girl relief. A profuse growth of papilloma was removed by direct laryngoscopy at this time and at frequent intervals during the succeeding several weeks. The tumor recurred so rapidly that therapy by X-radiation to the larynx was advised and administered according to the following schedule:

Date	Age	Port	Kv.	Filter	Dist.	M.A.	Time	Units
11/6	3 yrs.	50 sq. cm.	200	0.5 cu. lal.	50 cm.	15	9.2 min.	550 R.
11/7	3 yrs.	50 sq. cm.	200	0.5 cu. lal.	50 cm.	15	9.2 min.	550 R.

Total X-radiation = 1100 Roentgen units.

So far as is known the patient was not under a physician's care again until one of the present authors (T. E. W.) examined her eight years later (age, 11 years). The larynx was filled with papilloma, and the tracheotomy tube, which had been worn continuously since its insertion, provided her only airway. The papilloma was removed, but it was impossible to rid the patient of the tracheotomy. The girl was not seen again in this institution until she was admitted to the McMillan Hospital two years later (age, 13 years). The patient related that she suffered pain in the throat, associated with swelling and redness of the neck, for the preceding three days. Examination revealed a large mass of papilloma filling the hypopharynx and obstructing the glottic chink. An airway was being provided by a No. 1 tracheotomy tube which had been worn continuously. Redness and swelling of the neck extended from the level of the thyroid cartilage to the clavicle, and the neck was markedly tender to palpation. Therapy with penicillin resulted in regression of the inflammation. X-ray plates of the laryngeal region were made and the radiologist reported that "the thyroid and cricoid cartilages are not seen and the soft tissue lines are obliterated." A No. 3 tracheotomy tube was inserted on the fourth hospital day to increase the airway, and on the eighth day a No. 5 tube was inserted. Laryngoscopy was performed on the ninth hospital day, and repeated on the fourteenth, twenty-first, and twenty-fifth days, to attempt excision of the papilloma. After removal of tumor from the region of the true cords, the usual anatomic landmarks were obviously distorted and the arytenoids moved poorly, if at all. The tumor extended subglottically to the region of the tracheotomy. On the twenty-seventh hospital day a granulating fistulous tract developed just above and to the right of the tracheotomy tube. Purulent exudate drained from the tract for several days. Microscopic examination of a biopsy removed from the wall of the fistula revealed granulation tissue with subacute inflammation. The patient was discharged for a short interim, and was readmitted several days later as a candidate for laryngofissure. *This procedure was advocated with the hope that the papilloma* might be removed completely, and that, possibly, a skin graft might be applied to the larynx in order to construct a useful airway. On the ninth hospital day a midline incision was made through the external thyroid perichondrium. The thin and necrotic anterior halves of the thyroid cartilages were removed, and the fistulous tract (described above) was explored and found to extend through the cricoid-thyroid membrane. The entire subglottic area downward to the tracheotomy was filled with a papillomatous mass, which appeared to be infiltrating deeper tissues. Much of the tumor was removed, and the wound was packed with penicillin-gauze and left unsutured. Specimens submitted for histologic examination revealed irregular groups of atypical squamous cells deep within the lamina propria and adjacent to the remnants of cartilage. Owing to the observations made at opera tion and the studied opinions of several pathologists (discussed below), it was concluded that laryngectomy should be performed. Consequently, the larynx was resected 10 days later, and, except for the development of a small bit of tumor which was removed from the tracheotomy stome, the tumor has not recurred and the patient has been free from symptoms for almost three years.

COMPARATIVE PATHOLOGIC STUDIES AND INTERPRETATIONS.

Benign Papilloma. During the first 12 hospitalizations of D. M. (Case 1), which occurred between the ages of 16 months and three years, portions of the tumor in the larynx were submitted for histologic examination on five occasions. The third and fourth of this series of specimens were removed during the X-radiation therapy described in the schedule shown previously, and the fifth was removed approximately one month after the last treatment by X-radiation. The gross properties of the tumor in each of the five specimens, as observed by clinicians and the pathologist, were essentially identical with the others, and were characteristic of the usual benign papilloma of the larynx encountered in children. The histologic structures, likewise, were characteristic of this tumor and were essentially identical in all five instances, with the possible exception of slight variations noted in the specimens taken during and after the period of X-radiation. These variations are of indeterminable significance; they were regarded as unimportant.

During the first admission of V. S. (Case 2), when the girl was three years old, portions of tumor were examined histologically on one occasion, prior to X-radiation treatment. Sections of tumor removed during or after radiation therapy are not available. Initially, the tumor was grossly and microscopically characteristic of benign papilloma, and the laryngologist who first cared for the patient in the St. Louis Children's Hospital reported that there was no apparent change in the tumor following therapy with the relatively small amount of X-ray administered.

In general, microscopic examination of portions of tumor from both patients, at the times described above, revealed enormous "piling up" of proliferating squamous epithelial cells in a complex dendritic network, enclosing areas of well vascularized areolar connective tissue. The basement membrane was intact and regular, clearly delineating the proliferating epithelium from the supporting connective tissue, both subjacent to and within the mass of epithelial tissue. Slight variations noted in the tissue removed from D. M. (Case 1),

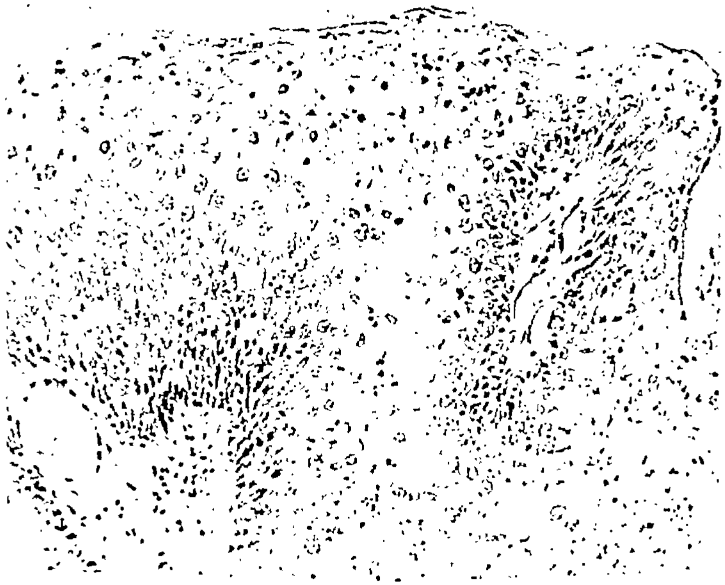

Fig. 1. Benign papilloma. Case 1, D. M. (biopsy at age, 35 months).

one month after X-radiation, consisted only of a few more mitotic figures in scattered regions, and, occasionally, small areas of squamous cells wherein the cytoplasm was vacuolated or the nuclei varied in size and staining reaction. Similar features were noted to somewhat lesser extent in tumor removed from V. S. (Case 2) prior to X-radiation. Figures 1 and 2, prepared from randomly selected areas of tumor from D. M. and V. S., respectively, illustrate the typical histologic structure of benign papilloma of the larynx. The tumors were closely similar in structure, and, in most areas, were essentially identical.

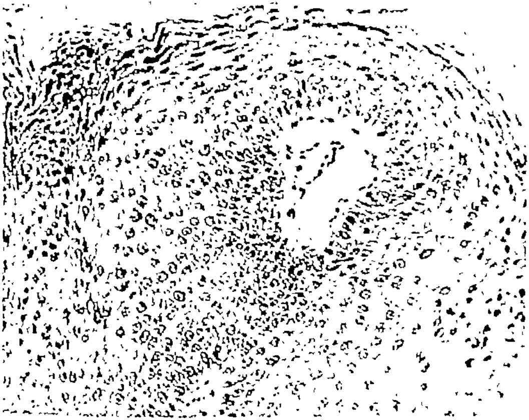

Fig. 2. Benign papilloma. Case 2, V. S. (biopsy at age, 36 months).

Phase of Transformation. For a period of more than eight years following the above studies, no histologic preparations of the tumor from D. M. (Case 1) are available for evaluation.* When the patient was almost 12 years old, he was this structure; furthermore, similar neoplastic cells were noted in the connective tissue between the first and second cartilaginous rings of the trachea (see Fig. 6, where the superior portion of the second ring is shown at the left of the photomicrograph). Careful study of the second interspace (inferior to the second ring), which was the lower extent of the excised tissue, revealed no involvement of this region by tumor. Although the cytologic features of the tumor were not as striking as they were in Case 1, the authors felt that a diagnosis of well differentiated epidermoid carcinoma was justified inasmuch as epithelial tumor cells were present in deep supportive structures, presenting a disorderly and unrestricted pattern of growth.

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*Reference to the clinical history indicates that the patient returned to the hospital for the fourteenth admission when seven years of age. According to the record papilloma was removed from the larynx at this time, but the tissue was not studied histologically.

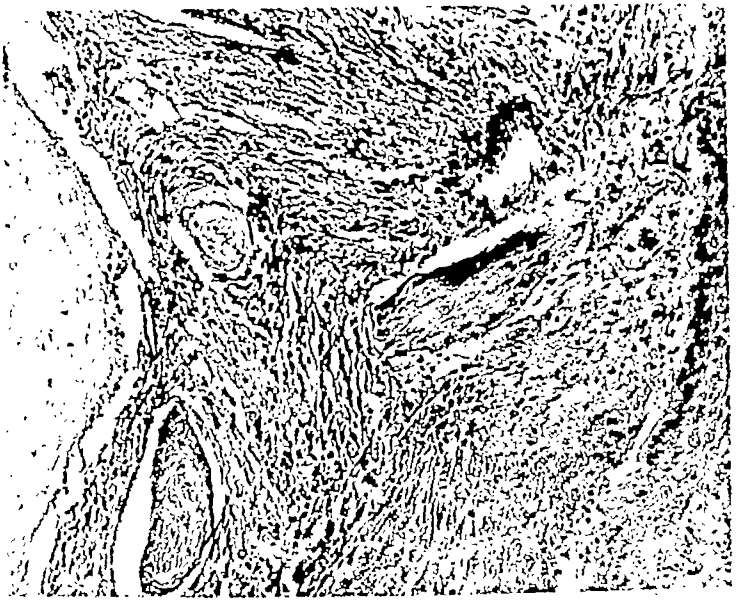

Fig. 6. Epidermoid carcinoma. Case 2, V. S. (laryngectomy specimen at age, 13 years).

COMMENT.

Jackson and Jackson[1] emphasize that papilloma in a child's larynx is "always benign and usually self-limited." Although laryngologists and pathologists agree generally with this thesis, the lesion is deserving of special consideration, nevertheless, because *a.* it is apt to cause serious obstruction to the passage of air, *b.* it sometimes regresses spontaneously, and yet *c.* it is prone to recur after removal, even after repeated removals in many instances. Perhaps a fourth reason should be added: despite the fact that papilloma of the larynx in a child is usually benign, there are rare instances in which a malignant tumor has developed within, or directly contiguous with, an area previously involved by papilloma of the "juvenile type." Papillomata in adults are occasionally the sites of development of carcinomas; many reports in the literature, as well as experiences in several clinics, affirm this. For example, Jackson and Jackson[2] reported that cancer of the larynx developed in 3 per cent of adults with papilloma studied in their clinic; in other series the incidence sometimes is slightly higher. In contrast, the present authors found only one paper reporting malignant transformation in papilloma of the larynx in a child. Unfortunately, the reference to this paper was lost, and subsequent searching failed to reveal the location of the paper; however, it is recalled that the patient was a child, about eight years old, with a relatively long history of recurrent papillomata of the larynx. Besides this case and the two described in this paper, only one other similar instance (a boy, 18 years old) has come to our attention.[3] So far as the authors are aware, this latter case has not been published.

Even a brief discourse on the pathogenesis of epidermoid carcinoma of the larynx in the two children described in this paper must be largely conjecture. In the first place, although some investigators *postulate* that the initial lesion (benign papilloma of the larynx in children) is a viral disease, the fact is that the causal and other factors responsible for this lesion are not known. Secondly, as a basis for further conjecture concerning cause and effect, certain features are common to the two cases reported herewith:

a. both children developed benign papilloma at relatively early ages;

b. both children suffered frequent and rapid recurrences of papilloma following skillful and apparently proper surgical removals; at no time was there any indication of spontaneous regression;

c. both children received X-radiation therapy to the larynx, relatively small in amount, at a time when the tumor was still histologically characteristic of benign papilloma;

d. owing to the extent of the disease both children were required to wear tracheotomy tubes for several years; in both instances, inadequate care in the long intervals between visits to the hospital must have a significant factor in the moderately severe inflammation which involved soft tissues of the neck and the tumor itself.

Thirdly, having harbored benign papilloma in the larynx for several years, both patients developed epidermoid carcinoma of the larynx; although both tumors were well differentiated, one showed a higher degree of anaplasia than the other.

The question of whether or not X-radiation played a rôle in the transition of the benign tumors into carcinoma is most difficult to evaluate, and our present knowledge is not sufficient to provide a conclusive answer. The X-radiation occurred several years prior to the development of the malignant tumor, and there are well recognized instances wherein carcinoma has developed long after an X-ray burn of the skin. In these cases, X-radiation was considerable in amount; in the two patients reported in this paper the dosage was relatively small. Three competent radiologists were consulted about this matter, and they stated rather dogmatically that on the basis of their experience the exposure to X-ray in these cases could have done no harm, especially the small amount given in Case 2. On the other hand it is interesting to note that two consulting pathologists who reviewed the slides from Case 1[4] raised this question independently of each other.

Another problem pertinent to the discussion is that regarding the rôle of the infection in each case. This complicating factor was present for a considerable time and was evident at the time when transition was noted in both tumors. Here again there is not sufficient factual knowledge to assess this factor adequately.

The causation of carcinoma is a complex and varied process. For example, Shope's papilloma virus is known to be the cause of a benign tumor occurring naturally in the skin of cotton-tail rabbits. Infectious papillomatosis may be transmitted serially in cotton-tail rabbits, but not all animals are susceptible and, in a large percentage of those in which lesions develop, spontaneous regression occurs. Malignant changes in the papillomata are unusual or rare. On the other hand, papillomata may develop in domestic rabbits, and, in a considerable number of these after some weeks or months, invasion of subjacent tissues and metastases to distant regions may occur; furthermore, the transformation to the malignant tumor may be brought about rapidly if a carcinogenic agent, such as tar, is applied to a virus-induced papilloma. Drawing on our knowledge concerning causation of certain malignant tumors in various organs, it is noteworthy that there probably is *not only one* causal agent of cancer, but that a *few* or *multiple* factors, previously present or coexistent in a given host, "set the stage" for the development of a malignant tumor. Owing to the lack of more specific knowledge, one is left with this somewhat unsatisfactory thesis concerning the development of carcinoma at, or within, the site of a benign tumor. From this one might speculate that the X-radiation, the infection, or a combination of both, perhaps in association with unrecognized factors (some of which may be inherent in specific strains or individuals), resulted in development of carcinoma at the site of previously benign papilloma of the larynx in the two children described in this paper.

SUMMARY.

1. Clinical and pathologic studies of a boy (age, 12 years) and a girl (age, 13 years) with epidermoid carcinoma of the larynx are presented and discussed.

2. Histologic studies of papillomata of the larynx in children should be performed thoroughly and carefully. In both these cases, at the time some pieces of tissue revealed malignant tumor, other portions were characteristic of the usual benign papilloma of the larynx. The distinctions could not be made grossly, but only after microscopic studies.

3. Whenever there is significant irregularity of cytologic structure or departure from the usual histologic pattern, the lesion should be regarded with suspicion.

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2. Jackson, C., and Jackson, C. L.: *Ibid.*, p. 373.

3. Children's Tumor Registry, American Academy of Pediatrics, Memorial Hospital, New York. Case S47-2208, Institute of Pathology, Rhode Island Hospital.

4. Unpublished correspondence from Dr. H. W. Dargeon, Children's Tumor Registry, to Dr. R. A. Moore, Washington University School of Medicine (through the courtesy of Dr. Moore).

EPISTAXIS — CASE REPORT.*

Report of a Case of Ligation of the External Carotid and Anterior Ethmoidal Arteries, and Section of the Posterior Ethmoidal Artery.

HARRY SACKADORF, M.D.,
Biloxi, Miss.

Although most episodes of epistaxis are easily controlled, a rare case may be encountered which proves intractable, requiring ligation of part or of all the blood supply to the affected naris. The case reported is that of a patient suffering from uncontrollable nosebleed, in which the entire blood supply to the affected naris required interruption.

REPORT OF THE CASE.

R. B., a 74-year-old white male with arteriosclerotic heart disease and positive Kahn, was admitted to the Veterans Administration Center, Biloxi, Miss., the morning of April 30, 1950, for treatment of severe nasal hemorrhage. He had been treated in this hospital for epistaxis from the right naris on two previous occasions, July, 1949, and March, 1950, when simple packing and one or two transfusions sufficed to control the bleeding. On neither occasion could any cause be ascertained aside from arteriosclerosis or latent syphilis.

The present episode of bleeding began at home April 28, 1950, when he suffered two mild nasal hemorrhages. These were repeated the following day. At 5:30 A. M., April 30, 1950, he developed severe epistaxis which his local physician could not control by packing. At 7:30 A. M., when he was admitted to the hospital, he was bleeding profusely from the right nostril and was in shock. Even the approximate origin of the bleeding could not be seen. Blood pressure was 120/80, blood count was RBC 3,200,000, hemoglobin 11 gm. and pulse 130. Both nares were quickly packed blood transfusion given, and morphine gr. one-quarter administered. Vitamins C and K, and penicillin, 500,000 units twice a day, were ordered. He bled repeatedly that day through anterior and posterior packings, so that two more transfusions were required to replace the blood loss. The following evening, May 1, 1950, because of continued severe bleeding, the right external carotid artery was ligated above the

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Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 12, 1950.

origin of the superior thyroid branch. This procedure had no effect upon the bleeding. Subsequent nasal examination, using suction to clear the naris, and direct nasopharyngoscopic investigation, showed the hemorrhage to be coming from the region of the right superior meatus, but no definite bleeding point could be seen. From May 1 to 17 he bled intermittently despite packing and frequent transfusions. On May 17, 1950, under intratracheal anesthesia, through a right medial periorbital incision, the periosteum was elevated from the orbital wall till the anterior ethmoidal vessels were encountered. These were doubly ligated and sectioned between ligatures. Further elevation of the periosteum posteriorly for about 1.5 cm. disclosed the posterior ethmoidal vessels. These were divided with a Freer's elevator; there was no bleeding from the sectioned ends. A loose gauze drain was inserted between the periosteum and orbital wall, and the periosteum was closed with catgut sutures above and below the drain. The skin incision was closed with cotton sutures. The following day the gauze drain and nasal packing were removed. The nasal hemorrhage had ceased, and there has been no recurrence since the operation.

The patient was given a total of 6,500 cc. of whole blood, vitamins B, C and K intramuscularly and orally, and adequate parenteral penicillin for its effect on the positive serology and for its preventive action against infection. Despite the antibiotic therapy, X-ray examination of the sinuses following surgery showed cloudiness of the right antrum attributable to the repeated packing. Two months later X-ray examination showed that the sinuses were clear.

DISCUSSION.

For practical purposes, one need only remember that the portion of the nasal interior below the middle turbinate is supplied by branches derived from the external carotid artery; the region above the middle turbinate by the anterior and posterior ethmoidal arteries which are branches of the ophthalmic artery, in turn a branch of the internal carotid.[1]

The clinical application of these facts is not so widely known as the anatomy. Many cases attest the effectiveness of ligature of the external carotid artery for refractory epistaxis;[2] but, where this procedure fails, even more modern textbooks[3,4] neglect to advise ligation of the ethmoidals. For example, both Jackson[5] and Boies[6] prefer ligation of the internal carotid, a suggestion which is invalid for two reasons: first, Dandy[7] demonstrated that serious cerebral damage may occur unless a favorable Matas[8] test be present, and preliminary partial ligation be performed; second, Weddell *et al.*[9] showed that ethmoidal artery bleeding cannot be checked by common or internal carotid ligation because of the competence of the circle of Willis with respect to the ophthalmic artery.

Management of cases such as this, which have also been reported by other authors,[10-12] should be guided by the principles (with certain additions) which Weddel *et al.*[9] evolved through trial and error in their own seven cases.

1. The patient is treated for shock due to blood loss.

2. Visualization following suction, and the effect of packing local areas in the nose, help to determine the source of bleding—whether above or below the middle turbinate. If manual compression of the common carotid fails to control the blooding, the ethmoidals are presumed to be implicated (anastomosis via circle of Willis).

3. If the bleeding be from below the middle turbinate, the external carotid artery should be ligated; if from above, both ethmoidals should be ligated primarily.

4. In either case, if bleeding continues following the primary ligation, the remaining blood supply to the affected naris should be interrupted.

5. Where the hemorrhage from one naris is so severe as to preclude determination of the source, the external carotid should be ligated first because its branches are most often involved.[3]

6. Where there is profuse hemorrhage from both nares, uncontrollable by any other means, one should adhere to the same schema even if it requires ligation of both external carotids and/or all the ethmoidals.

In the case reported, had it been possible to determine the source of bleeding prior to ligation of the external carotid, the procedure of choice would have been primary ligation of the ethmoidals.

SUMMARY.

1. A case of severe unilateral epistaxis is reported in which, ligation of the external carotid artery having failed to stop the hemorrhage, interruption of the ethmoidal arteries proved successful.

2. A logical procedure for determining and occluding the implicated arterial source is re-emphasized.

3. Where the source of bleeding is unknown, it may be necessary to interrupt the entire arterial supply to the affected naris.

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PLASTIC AND RECONSTRUCTIVE SURGERY.

SANFORD R. DIETRICH, M.D., Kansas City, Mo., and
JAMES BARRETT BROWN, M.D., F.A.C.S.,
St. Louis, Mo.

The literature of the past year deals almost entirely in civilian problems, with but few articles concerning wartime experiences.

NOSE.

The surgical repair of nasal deformities is summarized by Young.[1] Skin losses over the upper half of the nose are replaced with suitable free grafts, and pedicle flaps are used for covering the nasal tip and alar rims. Rhinophyma is a hypertrophic form of acne rosacea with an overgrowth of the sebaceous glands. The nose is cut down to normal contour and healing takes place from islands of epithelium that are left behind, or from the margins. Saddle nose is due to syphilis, trauma or too extensive a submucous resection. It is often necessary to reduce all dimensions of a humped or long nose. Retraction of the lower half of the nose may be corrected by moving the soft tissues forward and then inserting a cartilage graft to maintain this advancement. Distortions of nasal bones and cartilages by trauma and congenital deformities require repositioning of the framework and occasional cartilage transplants. Loss of lining of the nose may occur due to syphilis or to a burn, and repair with a skin graft is necessary. Partial or complete losses of the nose are best repaired with a forehead flap.

Nostril Border—A good nostril border, according to Brown and McDowell,[2] can be made from a composite ear graft (see Fig. 1). This graft contains skin and cartilage (see Fig. 2).

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 27, 1950.

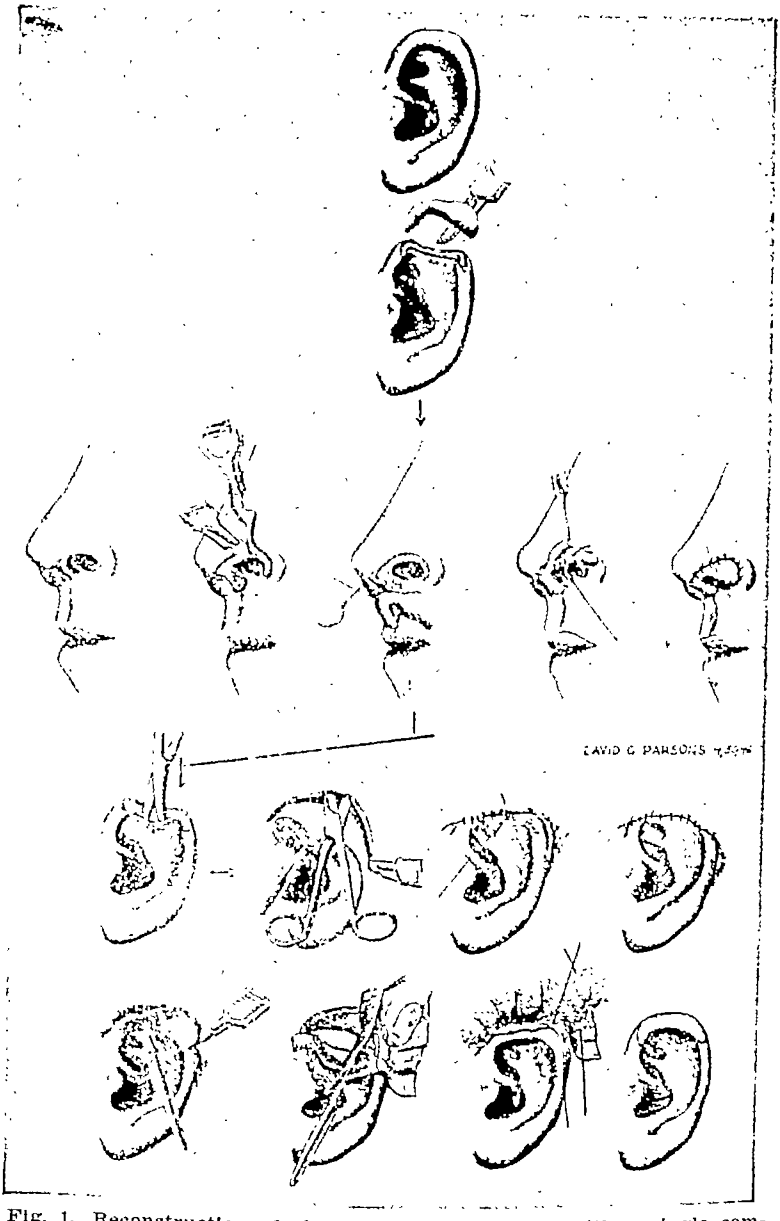

Fig. 1. Reconstruction of ala, angle and columella with a single composite free graft in one single operation. Secondary freeing of the ear from the scalp with a split graft, in this instance. The patient is also shown in Fig. 2. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

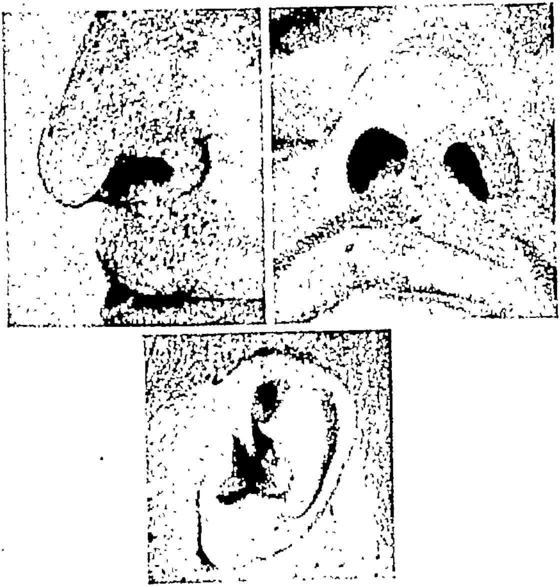

Fig. 2. Restoration of ala, angle and columella in one operation by a free composite graft from the ear. The ear is shown after later detachment from the head by a split graft. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

The nasal defect is prepared by cleanly excising the edges and then preparing a pattern of sheet lead or celluloid. An area is located on the helix of the ear having approximately the same curvature as the missing portion of the nostril border. The pattern is traced; and with a stab blade this portion is excised, taking care not to separate the parts of the composite graft from one another. The graft is sewed accurately into the nasal defect, and pressure is applied with internal packing and an external aluminum splint. The raw edge of the ear is buried under the postauricular skin for two weeks and later detached with a flap of this skin attached to it so that the flap may be rolled into a little tube to replace the missing portion.

Pelliciari[3] reports the reconstruction of the nasal tip with multiple composite free grafts.

Total Reconstruction—A pedicle-flap restoration of the nose is necessary if the whole feature or any considerable part of the framework is gone. Brown and McDowell[2] find that the best flap is one from the forehead, as this skin has better rigidity, color and texture for the repair, and contracts less than any other flap (see Figs. 3 and 4). A forehead at least 7 cm. high is essential. After delaying it twice, the flap is raised on a pedicle containing the supraorbital artery. The lining of the nose is made by turning the distal portion of the flap inward, and with local delayed flaps or a free split skin graft.

The "forehead rhinoplasty" is still the most popular method of nasal reconstruction according to Penn.[4] He describes a forehead flap based on the supraorbital vessels and raises the flap without a primary delay.

Structural Grafts to Nose—Brunner[5] reports on the fate of an autogenous-rib-cartilage transplant to the nose after 21 years. The cartilage in the dorsum nasi was broken up, and cartilage in the columella preserved its gross appearance. Denegerative changes of the cartilage were of minor importance as compared with invasion of the cartilage by connective tissue. Cartilage covered with perichondrium is more resistant to this invasion than cartilage without perichondrium.

Cottle and co-workers[6] report on the successful use of cancellous iliac bone grafts in nasal repair.

Seeley[7] prefers to use composite bone grafts of cortical and cancellous bone taken from the ilium to correct a saddle nose. Through alar-rim incisions the alar cartilages are dissected free, and the soft tissues are elevated over the dorsum. The "wedge bone graft" is then inserted, and bony union results. No absorption has been seen.

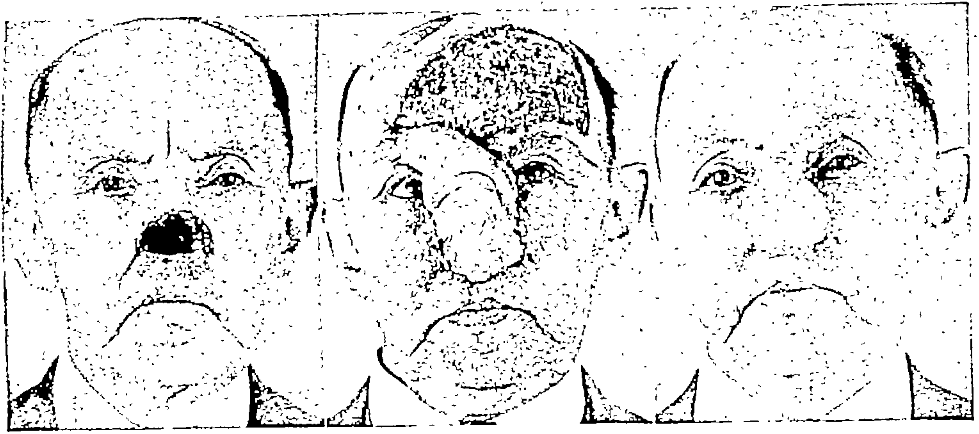

Fig. 3. Reconstruction of almost the entire nose by means of a forehead flap which was delayed in two stages. Complete lining of the new nose is essential in this type of "Indian" rhinoplasty and was obtained partly by turning down the skin from the bridge of the old nose and partly by turning the end of the forehead flap up into the new nostrils to meet it. The forehead was repaired by a split graft at the same time the pedicle was returned. L-shaped preserved rib cartilage graft inserted into the nose to provide skeletal support. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

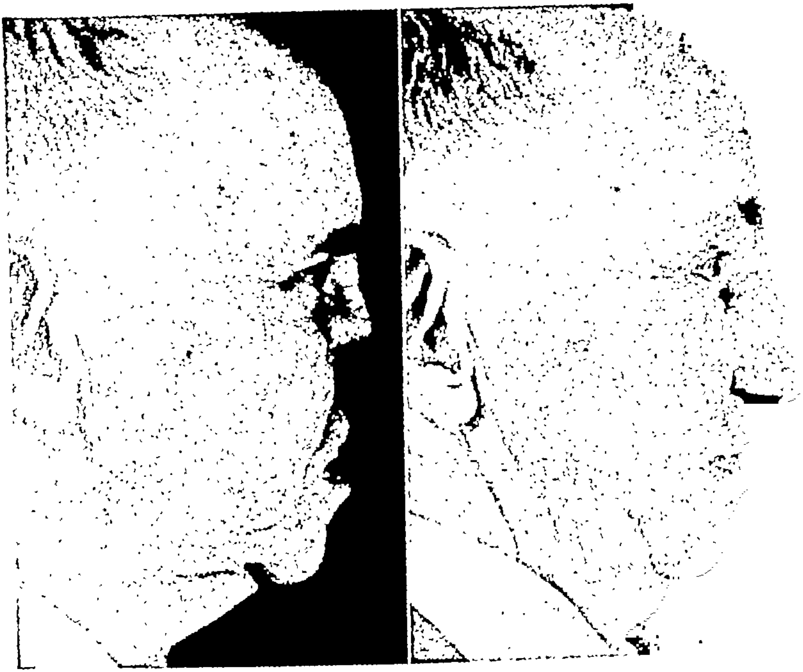

Fig. 4. Profile views of the patient in Fig. 3 before and after reconstruction. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

Fox[8] reports seven cases in which tantalum implants have been successfully employed in rhinoplastic procedures.

Columella—Gillies[9] discusses methods of repair for loss of the columella. A free composite ear graft is suitable if the septum is present and has a good blood supply. A flap may be used from the forehead, hand or arm. Bilateral nasolabial flaps may be used to reconstruct the columella. He describes a method, not before recorded, of using alar-margin flaps to make a columella.

Griesman[10] discusses "the mechanism of the suspension of the upper lip, because interference with this mechanism is the main danger of surgical treatment of the columella." The columella, nasolabial angle and the upper lip can be mobilized and separated by an intranasal technique. This method does not seem to disturb the shifting mechanism of the upper lip.

Cleft-Lip Nose—Huffman and Lierle[11] from clinical experience have gained the impression that the malformation of the nose associated with cleft lips is due not so much to inherent aberrations of the size and shape of the elements of the nasal skeleton (cartilaginous and bony) as it is to their being held in faulty positions. It is folly to try to build a straight nose on a crooked septum. If the septum lies to one side or the other of the midline, its replacement in a normal position is obligatory before the external nasal deformity is attacked.

Rhinophyma—Wehr[12] reports a case of rhinophyma treated by excision without skin grafting. The rhinophyma was shaved off with a razor.

Rhinoplasty—Marmor[13] feels that a corrective rhinoplasty, when complicated by obstructive deviation of the nasal septum, is better done in two stages, except in selected cases. McLaughlin[14] describes a case of early absence of the septal cartilage due to birth trauma. Restoration of the correct facial contour is done with dermal and bone grafts. Tausend[15] introduces an instrument he calls the "accurator." Its purpose is to assist in the trimming of the anterior border of the cartilaginous septum during a rhinoplasty.

EYEBROWS.

Eyebrows are important and can be replaced as described by Brown and McDowell[2] with strips of scalp transferred as free full-thickness grafts or as island flaps. Care should be taken in selecting the graft or flap so that the hair will be growing as nearly in the correct direction as possible. Free grafts are removed from the scalp just behind and above the ear and are cut through the entire thickness of the skin and subcutaneous fat. The graft is then inverted over an assistant's finger or on a table and each tiny grain of fat is dissected off with a fine thumb forceps and small iris scissors. When this is complete, the undersurface of the graft will be covered with the bulbs of the hair follicles like a fine brush. The graft is then sutured in place and a pressure dressing applied.

EYELIDS.

Brown and McDowell[2] correct ectropion of the eylids in one operation. Free grafts are put in place after the deformity has been freed by careful dissection (see Fig. 5). Most everted lids have sufficient mucosa to allow for protection of the globe if the skin covering can be obtained. The repair is made by carefully opening the junction of the everted mucosa and skin, turning the mucosa as a flap toward the globe, and then covering the whole raw surface with a thick split graft or full-thickness graft or a direct pedicle flap from forehead or temporal region (see Fig. 6). Full-thickness grafts from the supraclavicular area are best (see Fig. 7). The free graft may be held in place by using a local "stent" type of fixation with the long edge sutures tied over mechanic's waste and then an overall pressure dressing.

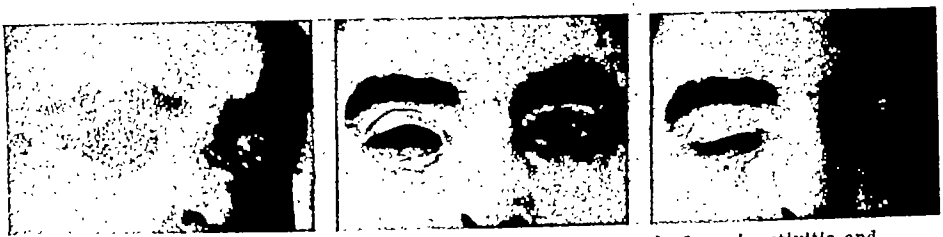

Fig. 5. Severe ectropion of all four lids with marked conjunctivitis and global irritation. Repair of all lids with thick split grafts, doing one set of lids (upper and lower) at each operation. Patient still needs a little more skin in the right canthus. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

Marcks[16] prefers to remove tumors about the eyelids with "scalpel surgery." He stresses the importance of a wide removal of the tumor and primary repair of the defect, for which he prefers a free skin graft. He does not attempt to discuss the secondary repair of the defects.

Straith and Lewis[17] offer the first report in the American literature of the Treacher Collins syndrome. This is an associated congenital deformity of the eyelids, ears and malar bones. The palpebral fissures are oblique and slant downward laterally. There is a deficiency of eyelashes from the middle portion of the lower eyelids, underdevelopment of malar bones and deformities of the ears with receding chin.

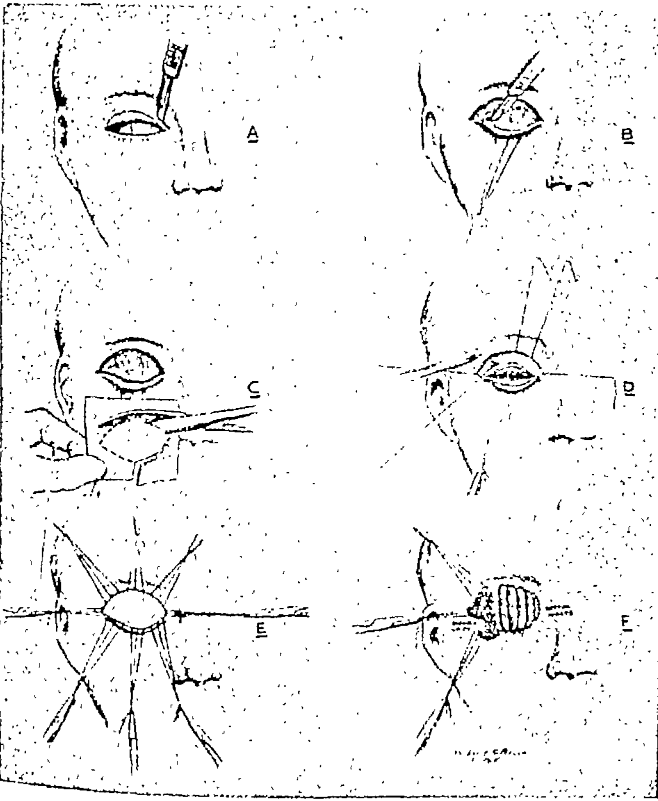

Fig. 4. Contracted area opened; surface and deep scar removed as necessary to allow a little more than normal expanse. Where pressure alone cannot be relied upon—as on the lids—the fixation is obtained and the proper opening of the area maintained by tying the long edge sutures over a pad of cotton waste, often first covering the graft with fine mesh grease gauze. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

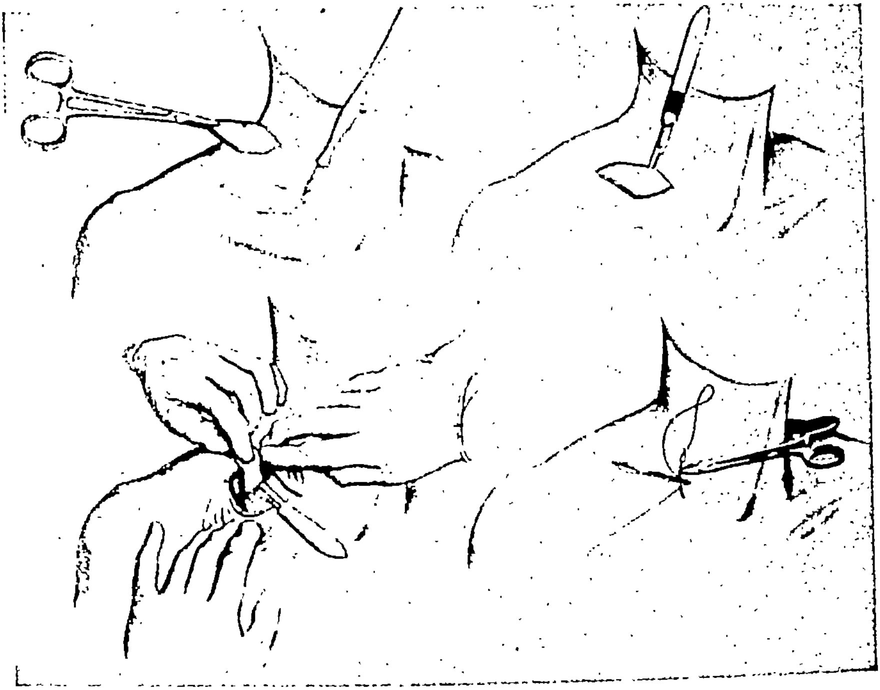

Fig. 7. Pattern of celluloid or other material traced on the neck with a pen and 5 per cent methylene blue. Assistants stretch the area taut to facilitate removal of the graft which is held with the fingers to avoid forceps marks. The wound is closed in layers with fine sutures. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

EYE.

Metallic imprints and integrated artificial eyes which provide an improved cosmetic appearance, both static and dynamic, have been developed by Hughes.[18] The integrated vitallium implants are partially buried in the orbit after the enucleation or evisceration of the eye. Some have been extruded.

Kiskadden, *et al.*,[19] report four cases of congenital anophthalmos successfully treated with acrylic forms of increasing size until an artificial eye of the size of the normal eye could be worn. The gradual dilatation of the conjunctival sac appears to help in the development of the orbit if begun at a very early age. The patient does not have to be old enough to cooperate. This method of treatment gives much more satisfactory results than surgical enlargement of the conjunctival sac with skin grafts.

EAR.

The total reconstruction of an ear can be done by Brown and McDowell[2] in as few as three operations (see Fig. 8).

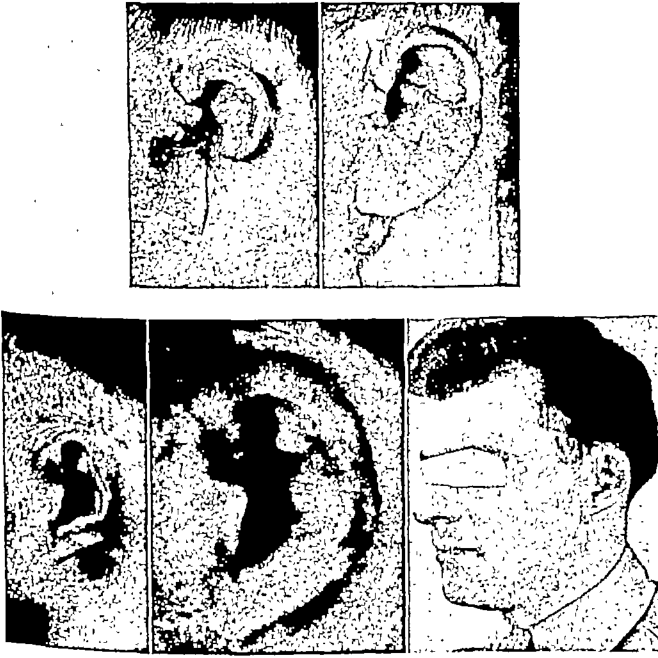

Fig. 8. Gunshot loss with occlusion of canal that was opened with flaps of local tissue. Reconstruction done with neck flaps brought up in stages with preserved cartilage support. (Bottom) Loss of about one-third of bulk from fire. Reconstruction with local flap preserved cartilage and split graft. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

Whatever is left of the stump of the ear is implanted under a scalp flap behind and above the ear. In two or three weeks when the union is adequate, a piece of costal cartilage is inserted under the scalp flap. In another three or four weeks the cartilage is dissected free of the skull so as to leave soft tissue attached to the undersurface of the cartilage and to the skull. The raw surface is covered with a single large thick split graft. A small tubed flap from the immediate neck region can be added for the helix if the patient desires.

An operative method to correct protruding ears is described by Becker[20] in some detail. The ear is set back to the head at the proper angle, and the postauricular sulcus is not obliterated, as in the procedures in which large sections of skin over the mastoid area and the auricle are removed.

Blake[21] has constructed seven ears with "prefabricated autogenous ear cartilages." The skin for the ear is supplied first by a tubed pedicle from the upper arm. To this is later added the supporting structure by a laminated sheet of cartilage shaped to a reasonable likness of a normal pinna by burial in the subcutaneous tissue of the abdomen between thin sheets of tantalum. A small piece of the eighth or ninth right costal cartilage is removed. The cartilage is cut into coarse flakes and spread and squeezed tightly between the plates of the tantalum sandwich or shaping cup and buried beneath the abdominal skin. The tantalum sandwich is removed three to seven months later and the laminated piece of cartilage inserted through an anterior incision into the soft tissue of the ear.

Wynn[22] has reconstructed an ear lobe as a one-stage procedure by wide undermining of the adjacent skin of the neck.

CLEFT LIP.

Le Mesurier[23] finds that in a complete unilateral cleft lip the cleft is pretty wide, although there is little real lack of tissue. The chief problem is to get sufficient tissue at the lowest edge of the lip to produce the desired pouting appearance. A quadrilateral flap from the cleft side of the lip is designed to fit on the medial side into a notch formed by the spreading apart of the two edges of a cut. A cupid's bow is formed. It is best to operate when the infant is three months old.

Steffensen[21] follows the same method of cleft-lip repair and reports uniformly good results. "Poor results have been due to the surgeon's inaccuracy in the finer details of approximation and not to the plan."

Dingman, *et al.*,[25] present evidence to show that blood loss is not excessive in the cleft-lip and cleft-palate operations if the usual methods for the production of hemostasis are followed. In bilateral cheilorrhapy, blood loss does approach the dangerous level. The addition of vasoconstricting agents even in very small concentrations is valuable in further reducing the blood loss.

Browne[18] discusses the elements of the deformity of the complete double-cleft lip. The failure of development of the columella is noted early; however, if the lip is closed, the columella will stretch out of itself. If the failure of development of the labiogingival sulcus is not remedied, the lip is immobile, the dental plate cannot be fitted and the muscle of the oral sphincter cannot be closed properly. The displacement forward of the premaxilla must be corrected by removal of the "prevomerine bone." The cleft of the lip should be closed with no skin of the lateral portions of the lip beneath the prolabium. The vermilion of the prolabium should not be used, as the muscles should meet and join under the skin of the prolabium.

Browne[19] sets back the premaxilla when the patient is three months old. The prevomerine bone is removed and the premaxilla set back and held in place with a special spiked plate and a suture through the alveolar ridge. The cleft alveolar ridge is closed at this operation. The second stage in the closure of the double-cleft lip is done four weeks after the first. The cheeks are undermined widely. The prolabium is converted into a "V," and the margins and vermilion are sutured to form a cupid's bow.

Huffman and Lierle[27] repair double clefts of the lip in three stages. First, the premaxilla is set back and the cleft of the hard palate closed. At the second operation, one side of the cleft lip is closed, and the other side at the third operation.

Three unusual clefts of the lips are illustrated by Braithwaite and Watson.[28] One patient had a cleft of the lower lip with agenesis of the mandible, eye and ear. Another showed a central cleft of the upper lip with a very small premaxilla and prolabium and a hole in the alveolar ridge only. Another presented a central notch of the upper lip.

As only five cases of macrostomia have been reported in Europe since 1881 and none in this country, Clifford[29] reports a case and its surgical correction.

Pickerill[30] has the mother nurse the infant patient undergoing surgery. This he feels has cut down on cross-infections. All dressings are done in the operating room with anesthesia. No one else will spend the necessary time and care that a mother will to care for her child during the postoperative period.

CLEFT PALATE.

Baxter and co-workers[31] write that the aim of surgery of the cleft palate is not only to close the defect but to establish normal speech. The latter objective is not obtained in many cases, owing to the anterior pull of the contracting raw surface on the nasal side of the mucoperiosteal flap, which results in a short palate. To prevent this, the authors apply a split thickness skin graft to the raw surface on the nasal side of the mucoperiosteal flaps. Three weeks later the cleft is closed and the palate set back. They now find it unnecessary to sever the greater palatine arteries to set back the palate. Palatal massage is begun four to six weeks after the operation. The authors feel that speech is markedly improved and that the palates are longer with their procedure.

Graber,[32] using mainly Roentgenologic means, has studied the bony face in cleft palate cases. He feels that early closure of the cleft palate will in some instances limit the growth potential of the maxilla denture. Limiting bands of scar tissue from the cleft-palate surgery may interfere with maxillary development. Slaughter and Brodie[33] also find that surgery of the cleft lip and palate, if poorly executed and poorly timed, can do more damage than good by inhibiting normal growth. This interference is directly proportional to the amount of injury to growth centers and to diminution of blood supply to the parts concerned. Other evidence is available to show that congenitally deformed parts, unless permanently damaged, grow at normal rates.

Trauma to the upper surface of the soft palate and opposing surface of the pharyngeal wall caused by operations for adenoids and tonsils may result in nasopharyngeal stenosis. Vaughn[34] corrects this by separating the soft palate from the pharyngeal wall and covering the raw opposing surfaces with local mucosal flaps.

MANDIBLE.

Prognathism—Waldron, *et al.*,[35] stress the fact that in the surgical correction of prognathism, it is the surgeon's responsibility to secure both cosmetic improvement and a maximum degree of restoration of dental function. Blind division of the ramus or of the condyle neck should not be attempted. There should be exact open-view control of both proximal and distal fragments after division of the mandible. With adequate preoperative study, effective splinting and immobilization, and adequate postoperative care, complications are rare when ramus osteotomy, retromolar resection or molar resection techniques are employed.

Reconstruction of Mandible—The techniques of mandibular reconstruction following nonunion of fracture or loss of bone originated during the first World War. Ivy, in 1920, reported a series of 103 cases. Between the two World Wars clinical experience with cancellous bone gave abundant evidence of its superiority to cortical bone. A survey by Blocker and Stout[36] of bone grafts to the lower jaw performed on service personnel in World War II shows that 1,010 grafts were performed. Sources of the graft were: ilium, 836; rib, 151; and tibia, 23.

Primary union occurred in 917 cases. There were 93 failures, of which 68 were successfully regrafted. The percentage of primary takes was 90.7 per cent and of final takes was 97 per cent.

A 25-year-cure of a giant-cell tumor of the mandible treated by currettement and X-ray is offered by Straith.[37] Five years after the removal of the tumor, a mortised iliac-bone graft was inserted for a nonunion of a fracture at the site of the removal. Functional and cosmetic result 20 years later is excellent.

Immediate reconstruction of the lower jaw following surgical excisions of large tumors has been accomplished by arino, *et al.*,[38] in two patients. The resection of tumor of the mandible is carried out in the usual fashion, making as large an opening in the buccal cavity as may be necessary. Cancellous bone chips are taken from the iliac bone. The oral mucosa is closed, and the bone chips are inserted into the wound from which the tumor has been removed. The masseter and internal pterygoid muscles are then sutured together under the lower border of the bone graft. The wound is closed and drained. An acrylic oral splint, external plaster splint, and interdental wiring are used for immobilization. Excellent functional and cosmetic results are reported in a case of an adamantinoma and of a sarcoma of the mandible. The sarcoma occurs postoperatively.

Adams[39] reports a case of bilateral hypertrophy of the masseter muscle. The surgical correction was accomplished by excision of a segment from the lower inner aspect of the muscle and removal of the protruding angles of the mandible.

Ivy[40] has corrected a case of benign hypertrophy of the mandibular condyle by surgical removal of the condyle. This was accomplished through a two-inch skin incision made beneath the left angle of the mandible. Part of the masseter muscle was reflected from the outer surface of the ascending ramus. A Blair curved pedicle needle was passed in the depths of the wound behind the neck of the condyle to emerge through the sigmoid notch beneath the zygomatic arch. A Gigli saw was passed around the neck of the condyle, and the neck was divided and the condyle removed. Functional and cosmetic result was excellent.

FACIAL INJURIES.

Immobilization of jaw fractures with internal wire pins is discussed by Brown, Fryer and McDowell.[41] When enough teeth are present, interdental wiring is the method of choice in the treatment of fractures of the mandible. When, for some reason, interdental wiring or other fixation would not be sufficient, the fractured lower jaw can be immobilized by a Kirschner wire, 0.05 to 0.08 inch, driven across the fracture site. Among the numerous indications for which this method has been frequently used are: lack of available teeth (see Fig. 9); symphysis fractures; fractures at the angle with the posterior fragment displaced into the upper buccal fornix (see Fig. 10); and when there is compounding into the mouth with infection and sloughing (see Fig. 11). In an edentulous mouth internal wires can be used in conjunction with circumferential wires around the patient's dentures (see Fig. 12). The wire should be driven across the fracture site with a power drill. Complications following the use of the internal wires have been slight, confined for the most part to slight drainage around the wires.

Mowlem[42] states that the most important causes of nonunion of the mandible are *1.* mechanical trauma and *2.* infection. Nonunion occurs in fractures of the body and symphysis and is rare in fractures of the condyle, angle and ascending ramus. Important factors that may contribute to nonunion are *1.* delay in fixation, *2.* inadequate reduction, *3.* inefficient fixation, *4.* tooth remnant or teeth in the fracture line, and *5.* gross contamination of the mandible. Before bone grafting can be performed for nonunion, *1.* all oral fistulas must be closed, *2.* all draining wounds must be eliminated for at least eight weeks, *3.* tooth fragments must be removed from the bone ends, and *4.* there must be an adequate means of immobilization.

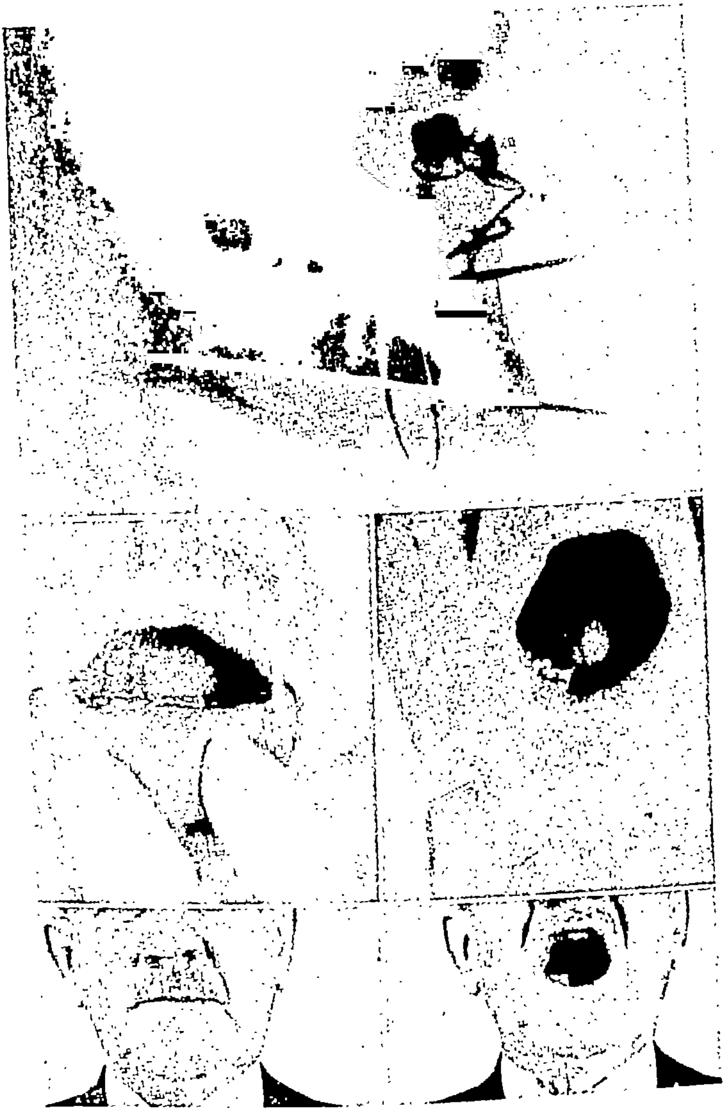

Fig. 9. Severe, splintered locally infected lower jaw widely compounded by anterior fragment being driven into the mouth, reduced and held immobilized until union was solid, by combination of internal wires, circumferential wire and arch constructed of twisted No. 24 steel wires. Normal opening action and ingestion of soft food possible during comfortable convalescence and on complete healing. (Brown, J. B.; Fryer, M., and McDowell, F. Plastic and Reconstructive Surgery, 4:31, Jan., 1949.)

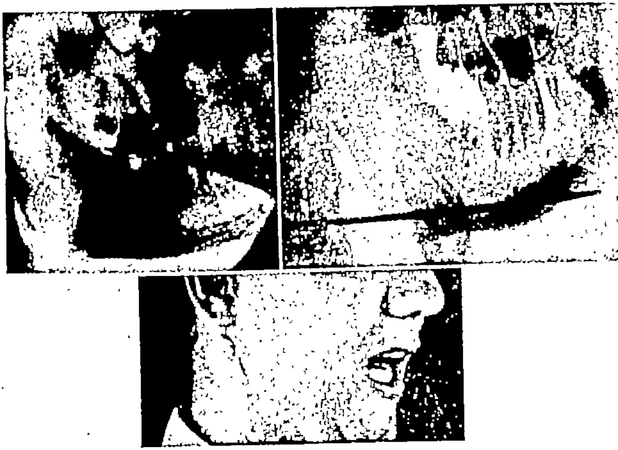

Fig. 10. Treatment of difficult fracture at angle without teeth to control posterior fragment, made simple and direct by internal wire driven through angle, across fracture site, into body of the jaw. Interdental wiring done because of associated fracture of the body of the mandible. (Brown, J. B.; McDowell, F., and Fryer, M.: Plastic and Reconstructive Surgery, 4:33, Jan., 1949.)

Cardwell[43] recommends reduction of fractures of the zygomatic tripod via an intranasal approach, using an appropriate male urethral sound. The operation can be done under local anesthesia. Gordon[44] describes a method for maintaining position in fractures of the malar bone in cases in which reduction is delayed for five or more days. The depressed portion of the malar bone is elevated. If the reduction cannot be maintained, a long bayonet skin needle is driven in through the malar to the nasal or nasal process of the maxilla. This gives good immobilization and maintains the reduction.

Moore and Ward[45] stress the importance of the early treatment of fractures of the facial bones. Severe deformities of the face may be due to untreated fractures of the face. Maxillary fractures may be classified as follows: *1.* horizontal fractures of the maxilla passing through the base of the nasal

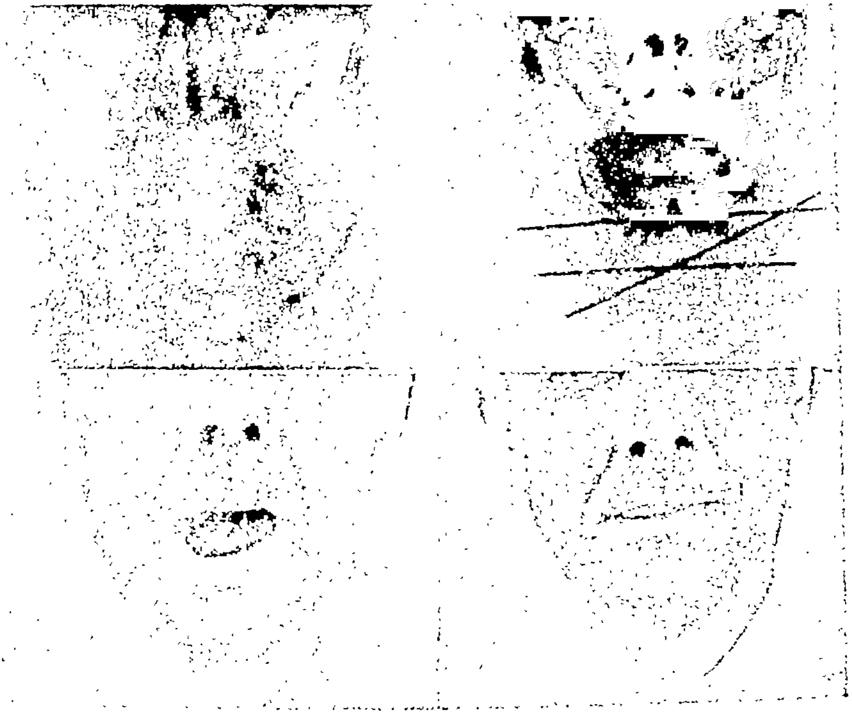

Fig. 11. (A) X-ray fails to show fine comminution of whole central segment of the lower jaw which felt like a "bag of salt," requiring, as shown in (B), three internal wires with twisted steel wire arch for definite immobilization. Local low grade infection with associated swelling of surrounding soft tissues and absence of usable teeth demanded the direct method of immobilization. (C) and (D) taken on removal of wires at eight weeks' time with normally opening, solid jaw. (Brown, J. B.; McDowell, F., and Fryer, M.: Plastic and Reconstructive Surgery, 1:33, Jan., 1949.)

and ethmoid regions, across the orbits, to the zygomatic arches; *2*. pyramidal fractures; *3*. low transverse or Guerin's fracture; *4*. vertical or unilateral maxillary fracture; *5*. depressed or comminuted fracture of the malar bone. If the fractures are not treated adequately early, they have healed in six weeks in malposition. Correction should be obtained not by camouflage methods only but by total refracture, reduction and immobilization of the fragments. By this method full correction of diplopia was possible in 22 of 26 cases.

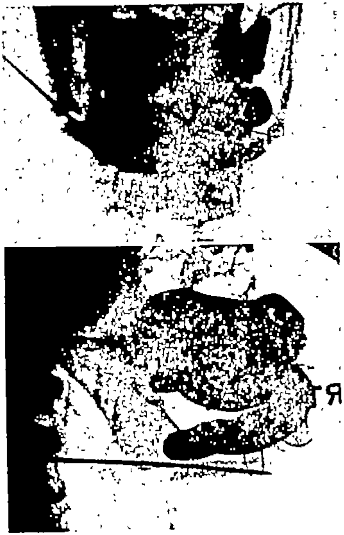

Fig. 12. "Pipe-stem" sized, odentulous lower jaw held steadily with the fracture in the body stabilized by circumferential wires around dentures and that at the angle immobilized by an internal wire, avoiding reduction. Healed solidly at eight weeks. (Brown, J. B.: McDowell, F., and Fryer, M.: Plastic and Reconstructive Surgery, 4:34, Jan., 1949.)

FACIAL REPAIRS.

The use of the tube pedicle in facial reconstruction is usually reserved for those cases lacking sufficient adjacent material for repair by other means. Steiss[16] finds the neck is a good donor site because of color match, the scar can be hidden and the blood supply is exceptional. Five cases of repair of facial defects with tube pedicles are reported.

Macomber[17] prefers to use autogenous bone grafts in correcting depressions of the forehead, nose and chin. Bone may be obtained from the crest of the ilium under local anesthesia. Cancellous bone is preferred, and cortical bone is used only when rigidity is at once required. The bone grafts become promptly fixed and retain their original dimensions when placed in vascular tissue, in contact with denuded bone or periosteum.

Dermal grafts have gained a definite place among the armamentarium of the plastic surgeon, for the reconstruction of facial contours. Schuessler and Steffanoff[18] state that dermal grafts tend to increase in volume in the early postoperative period, owing to the vascularization of the graft, and later on show a decrease from the original volume (approximately 15 per cent), owing to absorption. Of the 80 dermal grafts performed by the authors, only 7.5 per cent showed complications, and only 1.25 per cent of these showed epidermoid cyst formation. The hair-free areas of skin inside the thighs, on the abdomen and on the back offer the best donor sites for the dermal grafts. If the defect is so extensive that more than four layers of dermis must be used, then the correction should be carried out in stages.

Free fat grafts to the face for the repair of deformities is discussed by Stevenson.[40] Three cases with satisfactory results are reported. In these cases an attempt was made to implant an excess of about 50 per cent to allow for shrinkage.

Rubin[50] has successfully used polyisobutelene as a fat substitute in cheek deformities. Polyisobutelene is an artificial rubber. It is not as soft as fat, but it is well tolerated by the body and can be placed in the cheeks with no disturbance of the smiling mechanism. The body reaction to it is no greater than it would be to any inert foreign material as silk or nylon.

Polyethylene can be tooled to produce strings or "hairs" of the material. Teplitsky and Rubin[51] have successfully implanted masses of this polyethylene hair to fill out depressions of the face. The foreign-body reaction to it is slight.

Hemihypertrophy of the face is thought to be due to a defect of the germ plasm by Sutton.[52] A case and its surgical correction are presented. Weaver[53] reports the removal of tattoos of the face by excision and the use of sandpaper.

There are only nine reported successful primary repairs of severed parotid ducts, according to Sparkman.[54] He reports three additional cases. The anastomosis of the severed duct was done over a ureteral catheter. Multiple fine silk sutures which did not penetrate the duct lumen were used. The catheter was withdrawn at the conclusion of the anastomosis. A postoperative regimen which favored continued secretory activity of the gland was followed. No strictures were noted.

Bailey and Skaff[55] repair the severed parotid duct over a heavy thread which acts as a dowel. The dowel is anchored at each end with a lead shot. Five cases are reported.

NAEVUS.

Delario[56] presents an extensive classification of the common nonvascular naevi. He recommends prophylactic removal of naevi to prevent malignant degeneration if they are darkly pigmented, appear suddenly late in life, are subject to trauma, and naevi that show increasing pigmentation and size. Pack has reported a 14.7 per cent five-year survival in 267 patients with malignant melanoma treated with surgery.

ANGIOMA.

Brown and McDowell[9] write that port-wine stains should be considered as congenital anomalies of the capillary system in the derma, rather than as tumors. Some can be disguised successfully with cosmetics. They are almost uniformly insensitive to radiation in any form. Clean surgical excision with immediate free skin grafting is nearly always preferable to any destructive type of therapy. But the results are not perfect, and the problems should not be approached with great optimism (see Fig. 13).

Reidy[57] also stresses that capillary hemangiomas are insensitive to radiation. Port-wine stains in children may be treated with thorium. Excision and skin grafting of port-wine stains is the therapy of choice in others.

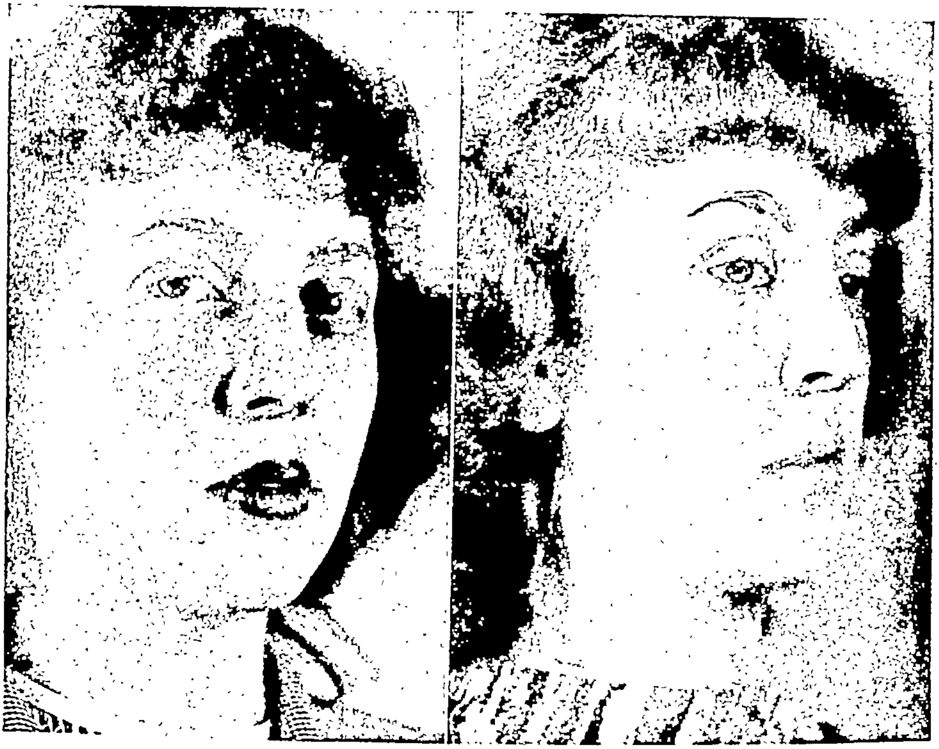

Fig. 13. Single operation repair of port wine stain. The hemangioma was dark and could not be covered satisfactorily with special cosmetics. Surgically excised and covered with free full-thickness skin graft. Result shown six months later. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

The dramatic effect of a cure of a cirsoid aneurysm treated by surgical excision is reported by Grimes and Freeman.[38] This aneurysm consists of two parts: the arteriovenous fistulas and the dilated afferent and efferent vessels. They are most frequently traumatic in origin and usually occur on the face, scalp, hands and feet.

There is still some question as to whether lymphangiomas are true tumors or congenital anomalies, but they do slowly increase in size and may become horribly disfiguring when present on the face. They are insensitive to irradiation. Brown and McDowell[2] prefer subcutaneous excision, if the overlying skin is not too warty or pigmented. At times, the skin is so ugly and the lesion so large that it is best to excise the whole thing and cover the area with a flap or graft.

MAMMAPLASTY.

Maliniac[59] states that the objectives of a mammaplasty should include esthetic form and conservation of function, as well as reduction in size. Preservation of blood supply, proper shaping of the gland and skin, and adequate mastoplexy are essential elements in all mammaplastic procedures. He describes a retromammary mammaplasty[60] which is a substitute for mammectomy with free grafting of the nipple, when the hypertrophy is mainly in the anteroposterior direction, and when it is desired to preserve the sensitivity of the nipple. The procedure consists of retromammary resections from the gland; the central segment, including the nipple, is preserved. This is followed by mastopexy by means of dermal loops and a double-flap skin plasty. In lesser hypertrophies transposition can be carried out in one stage. Mammaplasty with free grafting of the nipple should be performed only in true hyperplasias or in hypertrophies with advanced nodular cystic degeneration.

Bames[61] disagrees with Maliniac concerning the surgical treatment of gigantomastia. A one-stage mammaplasty with transposition of the nipple cannot be adequately and safely done on excessively large breasts. The excessively large breasts usually have little lactogenic function and, therefore, there is no reason not to do a one-stage mammaplasty with free transplant of the nipples. If a two-stage mammaplasty is planned to avoid free transplantation of the nipple, the patient frequently refuses the second operation.

Aufricht[62] corrects pendulous breasts at a one-stage procedure carried out simultaneously on both breasts with transposition of the nipple. He stresses the preoperative planning and feels that breast tissue can be excised in any area, as all breast tissue is well vascularized and it is not essential to avoid any particular branch of the mammary arterial tree. Lamont[63] describes a one-stage bilateral mammaplasty with free transplant of the nipple for mammoth breasts. The operation is carried out with the patient in the sitting position. Adams[64] has reconstructed the nipple by a free transplant of the labium minus. Brown[65] illustrates the construction of a bilateral one-piece latex breast prosthesis.

Foged[66] summarizes the operative treatment of abdominal-wall obesity in 35 patients. He recommends transverse excision of skin and fat between the umbilicus and the symphysis pubis.

FREE SKIN GRAFTING.

Baxter, *et al.*[67] and Thorek and Engel[68] have reported resurfacing of the penis with split skin grafts. Blanchart[69] has repaired an avulsion of the entire scalp and forehead with split thickness skin grafts. McCash[70] uses a plaster-of-Paris head cage in the fixation of free skin grafts to the head and face.

That contractures of the neck can be corrected with free skin grafts is well shown by Brown and McDowell.[2] If the repair is done before the area is healed, split grafts are always utilized. The deformity may be so great that the patient cannot look the world in the face and a wide open bite of the jaw may develop (see Fig. 14). The opening of the contracture is carried transversely completely across the neck and often out into normal skin on either side. All heavy dense scar is excised, and the remaining normal edge of tissue is dissected back to its normal position. Thick split grafts or full thickness grafts are used and fixed with a mechanic's-waste stent and a large pressure dressing.

Hypospadias repairs are effected by Young and Benjamin[71] in the preschool child with free inlay grafts. The chordee is corrected in one operation when the child is one year old. This is accomplished by complete exposure of the corpora cavernosa, excision of the fibrous remnants of the corpus spongiosum, the furrow in the glans, and often of part of Buck's fascia. The urethral orifice is transplanted laterally and backward, the halves of the glans and prepuce brought together, and the skin closed in zigzag fashion. The urethra can be

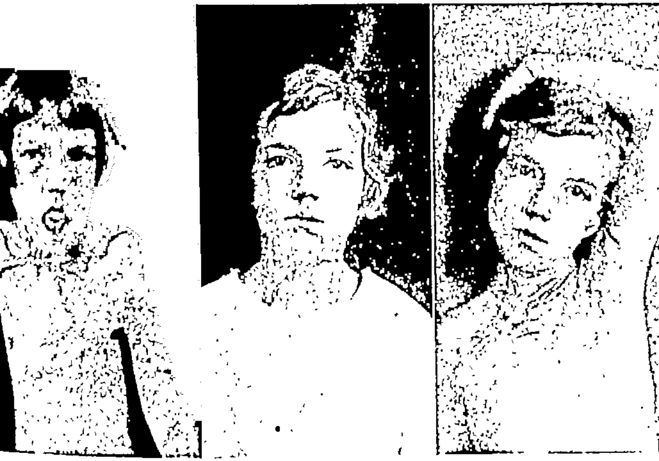

Fig. 11. Repair of contracted neck and axilla. Restoration was begun by completely releasing the neck deformity and covering the area with thick split grafts; at a second operation split grafts were used in the axilla, and later a full thickness graft was put across the front of the neck to give the final smooth appearance. (Brown, J. B., and McDowell, F.: Skin Grafting. Philadelphia, Pa.: J. B. Lippincott Co., 1949.)

reconstructed in a two- or three-year-old patient in one operation with a free inlay skin graft. A urethrostomy is performed high in the perineum to divert the urinary stream. A hairless dermatome graft is taken from the inner aspect of the arm for the inlay. The urethra is completed in one operation. There are certain advantages in this operation. The urethra has a normal exit in the glans penis. The urethra contains no hair. Multiple fistulas do not occur. No case of stricture is reported, and prolonged and frequent dilatation of the urethra has not been necessary.

Homografts of skin are discussed by Brown and McDowell.[2] These grafts may be lifesaving in certain instances. They take almost as well as autografts and may survive for from three to 10 weeks. During this time there is often a stimulus to epithelization so that complete healing may even occur after absorption of the grafts. This notable increase in wound healing has caused some observers to think that homografts survive permanently; but it is doubtful whether this ever occurs except in the instance of identical twins. McCoy[72] reports a case of homografting. Kearns and Reid[73] report two successively applied homotransplantations of split skin grafts from each parent to a burned boy. The grafts are reported present two years later.

The treatment of elephantiasis of the lower extremity is covered by Blocker.[74] This is a gravitational edema superimposed upon chronic obstruction of the lymphatics, aided and abetted by repeated bouts of acute infection. He attempts to excise as much diseased tissue as possible and replace it with healthy skin and guard against further gravitational edema by the use of supportive stockings. Following two weeks of bed rest, the skin and subcutaneous tissues are removed and replaced with skin grafts. Complete bed rest is enforced for 60 days.

By the topical application of dibromopropamidine (Mand. B. 1270) to wounds, surface infections caused by Proteus vulgaris and Pseudomonas aeruginosa can be eliminated, according to Champion and McDowell.[75] Clinical and experimental work by Localio, *et al.*,[76] seems to indicate that the administration of dl-methionine will assist in the healing of wounds in protein-depleted patients.

Robinson[77] has studied the blood loss from donor sites using the weighed-sponge method. It has been found that about 46 cc. of whole blood is lost per dermatome drum of skin removed. Straatsma[78] discusses the embryology of skin. Inexpensive replaceable blades for the Blair knife have been introduced by Brodenham.[79] The regeneration of sensation in grafted skin has been extensively studied by Hutchison, *et al.*[80]

PEDICLE FLAPS.

Brown, *et al.*,[81] stress the necessity of the cooperation of the plastic, orthopedic and neurologic surgeons in the restoration of major defects of the arm. Compound injuries of the arm that cause wide loss of soft tissues and bone leave crippling defects. Healing is by dense, avascular fibrous tissue. The scar epithelium is unsatisfactory and repeatedly ulcerates. To obtain relief from this binding, distorting scar, with surface ulceration and deep fixation of bone, joint, tendons and nerves, there needs to be first removal of the scar and replacement of surrounding normal soft parts and bone to approximately their normal positions. The surface defect can then almost always be covered by an immediate short broad pedicle flap from chest or abdomen. Within 14 to 21 days the short broad pedicle can be severed and the surface coverage completed. After several weeks, work on nerves, bone and tendons can be done through adequate surface coverage.

Cannon and Trott[82] emphasize the value of direct flaps in extremity repairs. The reconstruction can be hastened and simplified without jeopardizing the result. Remote flaps can be completed in two operations about four weeks apart with a sufficiently broad pedicle. Delays in detaching these broad flaps are necessary only if the pedicle itself is to be used in the resurfacing.

Conway, Stack and Docktor[83] from work on experimental animals conclude that both the saline-wheal test and the fluorescein test give more accurate evaluation of the circulatory efficiency of a tubed pedicle than do clinical observations alone. Cuthbert[84] describes a "marsupial" skin flap to cover large rectangular skin defects. Five operative procedures are necessary.

Hynes, *et al.*,[85] use a fluorescein test for determination of the blood supply of tubed pedicles. Ten cc. of a 5 per cent solution of fluorescein are injected intravenously. A bowel clamp is placed across the end of the tube to be divided. A series of intradermal wheals is raised at intervals of 2 to 4 cm. along the length of the tube with 1:1000 histamine phosphate. Under ultraviolet light the progression of the fluorescence along the tube is observed. Using this test and the atropine-absorption test, Hynes[86] is able to effect the rapid transfer of abdominal tubed pedicles to the lower extremities. In suitable cases abdominal tubed pedicles have been transferred to the leg in three instead of four stages.

DECUBITUS ULCERS.

A report of 334 operations for closure of decubitus ulcers in paraplegics has been prepared by Blocksma, *et al.*[87] As most of those patients were ambulatory and sitting several hours each day, 178 of the operations were for ischial decubiti. Sulfasuxidine was used preoperatively and postoperatively. The tendency has developed to become more radical in the excision of the decubitus ulcer and the underlying bone. The underlying bone was found diseased in 75 per cent of the specimens. Sacral decubiti were best repaired by local pedicle flaps to fill the defect after excision of ulcer and projecting bony spine. After excision of trochanteric decubitus and the greater trochanter, an anterior thigh flap is used to fill the defect. For an ischial decubius the ischial tuberosity is removed with Gigli saws. Battle[88] has also written on decubitus ulcers in paraplegic patients.

BURNS.

Wallace[89] presents a preliminary report on a series of superficial burns of moderate extent in babies and children treated systemically with penicillin, locally with penicillin powder, suspension immobilization and exposure to the air. Braithwaite[90] feels that plasma is satisfactory therapy of shock in patients with third degree burns covering an area not greater than 20 per cent of the body surface. Whole-blood transfusions should be used if the third-degree area is greater than 20 per cent. Eisenstodt[91] recommends that in the moribund burn patient the granulations not be removed at the time of skin grafting. He finds that thin split grafts will take with a hemoglobin as low as 30 per cent and a plasma protein level of five, and in the presence of infection Owens[92] directs attention to the presence of Sudeck's osteoporosis following first and second degree burns.

Braithwaite and Beales,[93] following the concepts of Perry and Shaw (1894), conclude that Curling's ulcers will become less frequent in burns as penicillin can be used to control the infection in the burn patient.

CHRONIC ULCERS.

Byars and Letterman[94] write that infection, general debilitation, hypoproteinemia, anemia and chronic shock occur as complications to chronic ulcerations. The only final satisfactory solution to the ulcer problem is to attain a well-healed wound at the earliest possible moment. The application of topical agents alone to a wound will not achieve healing. Large areas of skin loss by avulsion may be replaced immediately. The use of split thickness skin grafts is the most satisfactory solution to the chronic ulcer.

Douglas[95] suggests an explanation of the nonhealing traumatic ulcer. He has found striated muscle fibres in the scar in the base of these indolent ulcers. The enclosed muscle fibres produce an excess of fibrous tissue, the immediate cause of the nonhealing process. This disaster can be prevented by great care in the toilet of all operations, care being taken to see that the muscle sheaths are closed.

RADIATION BURNS.

Brown, McDowell and Fryer[96] emphasize that excessive exposure of the skin to radiation may produce changes in the skin that will require excision and repair. The changes are most commonly seen on physicians' hands due to exposure during fluoroscopy (see Fig. 15). Radiation burns occur on dentists' fingers (see Fig. 16) and on patients due to long fluoroscopic examination. These burns also result from excessive radiation treatment given acne (see Fig. 17), eczema, port-wine stains, epidermophytosis, pruritus ani and superfluous hair (see Fig. 18).

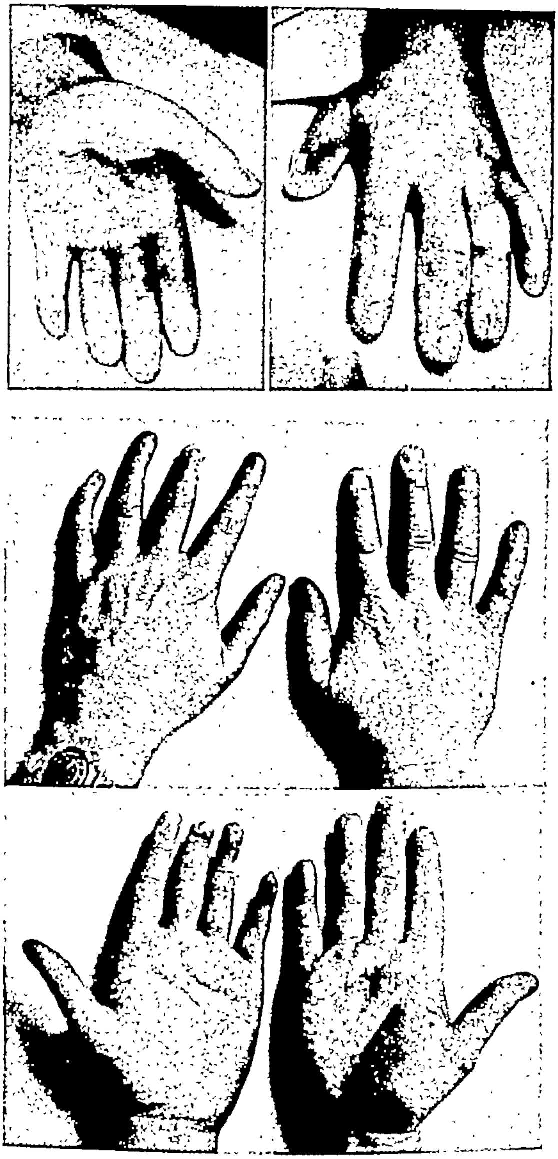

Fig. 15. Flouroscopic burns of doctor. (a, b) Acute burns on both hands—left worse than right. Patient required 6 gr. of morphine a day. Patient came in with consideration of amputation of hand. (c, d) Result of general protective care and early resection and free grafting. Recovery was such that he was able to follow a full career in anesthesia, whereas he formerly was a surgeon. (Brown, J. B.; McDowell, F., and Fryer, M. P.: Surg., Gynec. and Obst., 88:609, May, 1949.)

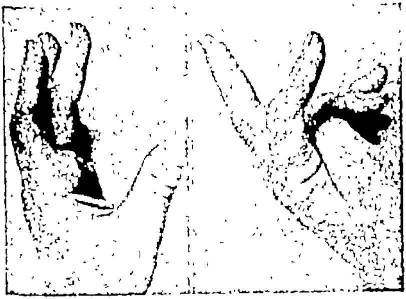

Fig. 16. Dentist's burns. Typical burn of dentist's fingers from holding films in patients' mouths. Resection with immediate split graft. One operation. (Brown, J. B.; McDowell, F., and Fryer, M. P.: Surg., Gynec. and Obst., 88:609, May, 1949.)

The pathologic process includes *1.* loss of skin appendages, *2.* replacement of normal collagen by dense acellular hyaline collagen, *3.* an obliteration of blood vessels of the corium, *4.* necrosis in the coremium about thrombosed telangiectases, *5.* coal spots, and *6.* carcinoma. Generally, telangiectases occur in three to 10 years, coal spots and keratosis in 10 to 15 years, and carcinoma in 20 to 25 years.

Severe acute radiation burns are very painful. They are less painful if covered with a bland ointment, a loose cotton dressing, and immobilized and elevated. The diagnosis of the chronic radiation burn is often overlooked. The triad of atrophy, telangiectasis and keratosis of the skin should lead to the diagnosis. The progress of the change to carcinoma is inevitable if the patient lives long enough. The excision and repair should be done before ulceration, pain and malignant change have occurred. Following excision of the irradiated skin the following variation in treatment may be useful sometimes: *1.* the wound is left open to be skin-grafted later, *2.* the wound may be dressed in a split graft and the patient advised that it may have to be repeated, *3.* immediate closure of the defect with adjacent or distant flaps, *4.* delayed closure

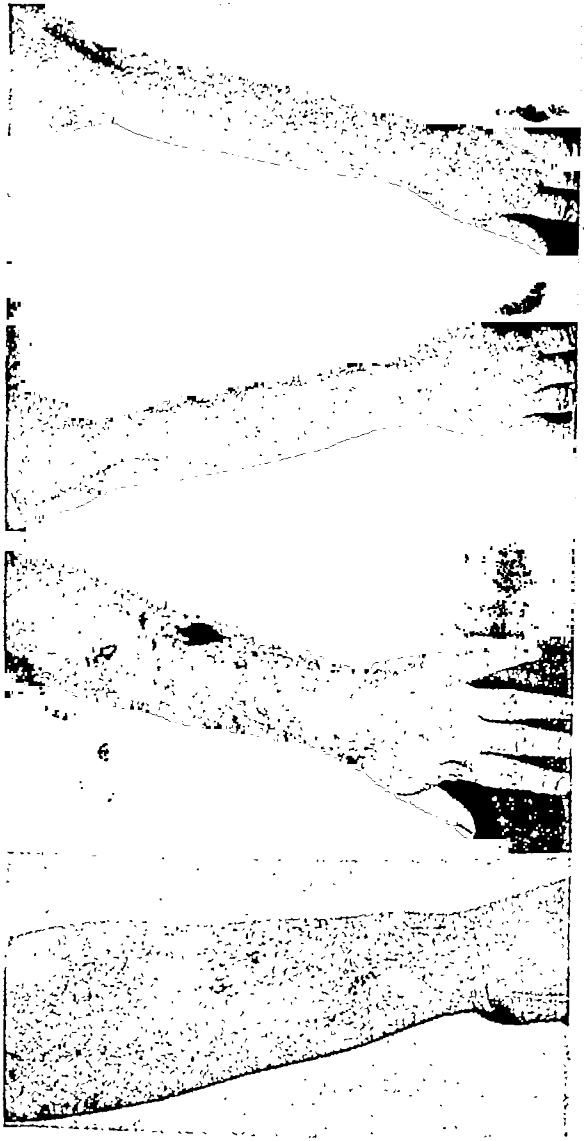

Fig. 17. Commercial hair remover burn. Excessive burning from commercial X-ray treatment to remove hair. Patient's arms, axillae and legs were completely burned. Treatment consisted of excision, in two stages of all skin of lower arm and immediate split grafting. (Brown, J. B.; McDowell, F., and Fryer, M. P.: Surg., Gynec. and Obst., 88:609, May, 1949.)

with pedicle flaps, or *5.* if bone or tendon is exposed, the use of pedicle flaps. There are two essentials: *1.* to get rid of the lesion, and *2.* to repair the area.

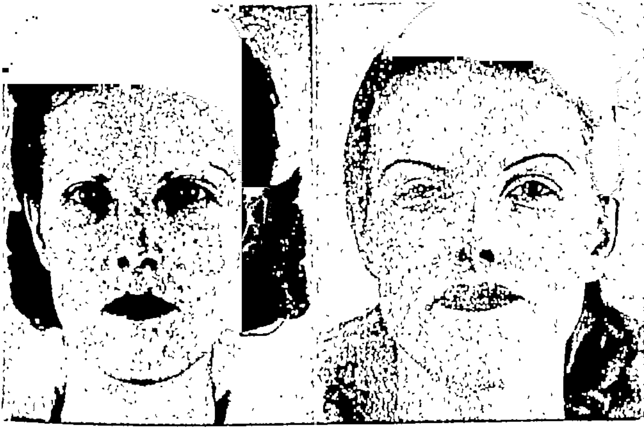

Fig. 18. Widespread involvement following treatment for acne. As usual the tip of the nose, being closest to the tube, is the worst burned. Repair by resection and immediate split grafting. Four operations were necessary to repair both cheeks, whole nose, upper lip. (Brown, J. B.; McDowell, F., and Fryer, M. P.: Surg., Gynec. and Obst., 88:609, May, 1949.)

Brown, McDowell and Fryer[97] assure us that atomic-radiation lesions may increase as work in turning mass into energy increases. The lesion produced by atomic radiation is not a new one entirely, but the time element of the development is faster owing to excessive exposure in a short space of time. The burns probably occur from both beta and gamma radiation. Conservative therapy, especially about the hands, avoiding early amputation, is indicated because hopeless looking fingers may be spared.

Ivy[98] reports on the repair of full-thickness cheek defects following irradiation necrosis. Cronin[99] pleas for a greater recognition of the potential dangers of radiation therapy in both benign and malignant conditions. Any measure short of extirpation of the lesion can be only palliative. Freeman[100] advises using fluorescein as an agent for determining the extent of deep radionecrotic lesions.

HAND.

Brown and McDowell[2] have found that the most common form of syndactylism is symmetrical webbing in two normal hands between the third and fourth fingers throughout their length. The operation is best delayed on these until the patient is three years old. The fingers are divided, being careful at the proximal end of the new fissure to avoid injury to any of the proper vessels or nerves. The resulting raw surface is covered with a single full-thickness skin graft. It is best to avoid operating on both sides of any one finger at one time. Sodenberg[101] reports a case of syndactylism associated with absence of the pectoral muscle.

A method for restoring an effective grasping function to a hand crippled by the loss of all five digits is presented by Cannon, Graham and Brown.[102] Elongation of the thumb and clefting of the hand are accomplished without disturbing normal sensation in the hand. The presence of movement at the articulation of the first metacarpal with the greater multangular is essential. The thumb is elongated if the bony stump is shorter than that of the index finger. This is accomplished through a dorsal incision, and the head of the second metacarpal is transplanted to the end of the first. Kirschner-wire fixation is used. The base of the second metacarpal is left to prevent collapse of the metacarpal arch. The cleft is made using a dorsal rectangular flap across the base of the cleft and thick split skin grafts to the sides.

The restoration of pinch function to the crippled hand by reconstruction of an opposition digit was the most common need found by Baxter, *et al.*[103] Reconstruction of the thumb can be done in many ways. They prefer to lengthen the thumb with a tube pedicle and bone graft in four operations.

Evans[104] discusses major injuries of the hand and emphasizes the importance of immediate and complete skin coverage to prevent infection, fibrosis and limitation of function. Hyroop[105] has transferred a metacarpal with or without its digit in 34 cases. This has proved an effective and efficient method of repair of certain types of crippled hands.

In the normal hand, notes Young,[106] the skin of the thumb-index web, thenar eminence and midpalm is a functioning unit which allows the thumb to move almost as a universal joint. Scar contracture of this region can be relieved by one of four methods: *1.* Z-plasty, *2.* local flaps, *3.* free skin grafts, or *4.* thinned pedicle-flap transfers.

Braithwaite and Watson[107] advise the early grafting of burns of the hand. Early excision of full thickness of damaged skin down to and including the deep fascia should be performed and followed by skin grafting. Braithwaite, *et al.*,[108] discuss the anatomy and function of the extensor-tendon complex of the hand.

Bruner[109] recommends the use of a dorsal flap to cover the palmar defect after excision of skin and fascia for an advanced Dupuytren's contracture. Flynn[110] discusses flexor-tendon grafts to the hand. Patton and Strain[111] use a molded acrylic splint for a palm and finger graft inserted to correct a burn contracture. The split was used from the twenty-fifth to the ninetieth postoperative day.

NEOPLASMS.

Skin—New[112] believes that cancer of the face does not receive as efficient therapy as one would expect from the site of the lesion and the facility with which a diagnosis can be made. The biopsy should include the entire cancer if possible.

Kraissl and Conway[113] show that Langer's lines are not the most suitable for planning the direction of incisions on the face. The normal wrinkle lines are at right angles to the action of the muscles of expression and afford the best guides to incisions on the face.

Evidence from 40 cases of malignant melanoma treated by surgical excision of regional lymph nodes by McCune[114] shows the value of early surgery. The general principles for treatment of the malignant disease of the face and scalp laid down by Fitz Gibbon and Bodenham[115] are: *1.* wide excision with knife and not diathermy, *2.* removal of regional lymph nodes if palpable, and *3.* immediate coverage with free skin grafts

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THE GRADUATE SCHOOL OF MEDICINE OF THE UNIVERSITY OF FLORIDA ANNOUNCES MID-WINTER SEMINAR IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Fifth Annual University of Florida Mid-Winter Seminar will be held at the Sans Souci Hotel, Miama Beach, Fla., beginning on Jan. 15 and continuing through Jan. 20, 1951. Lectures on Otolaryngology, Jan. 15, 16; on Ophthalmology, Jan. 18, 19, 20. On Jan. 17, a program will be presented by the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc. The combined meeting of the Mid-Winter Convention of the Florida Society of Ophthalmology and Otolaryngology and the Seminar will be held Wednesday *evening*, Jan. 17, 1951.

The complete schedule of lectures and reservation blanks for the course will be mailed to all E., E., N. and T. specialists early in September. For those who desire to register immediately, a check for $10.00 payable to "The University of Florida Mid-Winter Seminar" must accompany your application for registration. This deposit is not returnable. The balance of the course fee is payable at the registration desk at the Seminar headquarters—on your arrival.

The Division of Ophthalmology is under the direction of Shaler Richardson, M.D., Jacksonville; Nelson M. Black, Miami; Charles Boyd, M.D., Jacksonville; Bascom Palmer, M. D., Miami.

The Division of Otolaryngology is under the direction of H. Marshall Taylor, M.D., Jacksonville; Walter T. Hotchkiss, M.D., Miami Beach.

Application for registration should be sent to Dr. Walter T. Hotchkiss, 541 Lincoln Road, Miami Beach 39, Fla.

Registration fee for the entire course is $40.00. A group of America's outstanding eye, ear, nose and throat specialists will participate in the program.

This 1951 session will be held at the beautiful Sans Souci Hotel on the ocean front. Here you can combine a wonderful

Winter vacation and a highly instructive schedule of lectures. The hotel is equipped with pool and cabanas, excellent dining room and coffee shop and offers every modern facility of an outstanding hotel in America's Wonderful Winter Playground.

Special rates of $18.00 (double). For those seeking more moderate accommodations, the Scott-Bryan directly opposite the Sans Souci offers rooms for $12.00 single or $14.00 double. The El Morocco, also opposite, offers excellent accommodations for $10.00 single or $12.00 double. Ocean front facilities at the Sans Souci are available to guests of the Scott-Bryan and El Morocco Hotels. Make your reservation as early as possible, directly with the Sans Souci Hotel, which has charge of all reservations.

BOOK REVIEWS.

Your Deaf Child. A Guide for Parents. By Herbert R. Myklebust, Ed.D., Professor of Audiology, Northwestern University, Evanston, Ill., with foreword by Hallowell Davis, M.D., Director of Research, Central Institute for the Deaf, St. Louis, Mo. 132 pages and 17 illustrations. Springfield, Ill.: Charles C. Thomas, 301-327 East Lawrence Avenue, 1950. Price $2.50.

This is an excellent book written entirely for laymen and particularly for parents of deaf children. It contains a fund of information in a manner easily understood and in good, clear English. The chapters on the parent's attitude toward the child and the training of the deaf child in its younger years are particularly valuable. Otologists would be well advised to read this book for their own information and to advise the parents of their young deaf patients to get the book for a handy reference at home.

A very valuable addition is Chapter Eight in which Dr. Myklebust lists organizations which are interested in the deaf and hard-of-hearing children and books and periodicals which might help the parents of these children and also a list of the schools for the deaf and hard of hearing in Canada and the United States. This chapter alone would be worthwhile having in an otologist's office.

All in all, this is an excellent book which one can heartily recommend to otologists and to the parents of deaf children.

Bronchoesophagology. By Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., Honorary Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia; and Chevalier L. Jackson, M.D., Sc.D., F.A.C.S., Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia. 366 pages with index and 261 illustrations. Philadelphia: W. B. Saunders Co., 1950. Price $12.50.

This book is, in effect, a new edition of the Jacksons' previous books on bronchoesophagology. All the material is brought up to date, it is profusely and beautifully illustrated and is a book, I believe, essential for all students of ear, nose and throat.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM; Model M-35; Model P-15.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Southern Section: Miami, Fla., Jan. 17, 1951.

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Basic Sciences in Otolaryngology
September through June

THE

LARYNGOSCOPE.

VOL. LX DECEMBER, 1950. No. 12

CLOSURE AND REVISION FOLLOWING THE FENESTRATION OPERATION.*

H. DAVIS, M.D.; T. E. WALSH, M.D.,
and E. ELDERT, M.A.,
St. Louis, Mo.

INTRODUCTION.

The two chief problems in the prognosis of the fenestration operation for clinical otosclerosis are: first, what will be the immediate gain for speech, and second, will the improvement be permanent. The first question we have analyzed recently[1] on the basis of a long-term study of the patients fenestrated by one of us (T. E. W.) at McMillan Hospital (Washington University). The present study deals with the second question and reports the incidence of closure in the same series of operations; it also includes our very disappointing experience with the "revision" operation in which the surgeon reopens a fenestra that has been closed by the regrowth of bone.

SURGICAL PROCEDURES.

Before October 28, 1946, the cartilage stopple[2] was routinely employed in the fenestration operation, following the

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*This study was begun under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under Contract V1001M-577 between Veterans Administration and Central Institute for the Deaf. The routine testing and the collection of data were later transferred to the Hofheimer Audiology Laboratory at McMillan Hospital. The work there is supported by a grant from the Hofheimer Foundation.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Sept. 28, 1950.

mistaken idea that it tended to prevent the normal regrowth of bone and consequent closure. When it became apparent that this was emphatically not the case' the stopple was abandoned. The cases in which the stopple was employed are not included in the present study, but a few early cases in which the stopple was omitted are fortunately available now (April, 1950), four or five years after operation.

Although there have been modifications from time to time in the incisions, in the extent and technique of mastoidectomy and in the pre- and post-operative care of the patient, the technique for creation of the fenestra has been constant. Until March 14, 1947 (Case No. 185), a Cameron loupe was used for magnification. From Case No. 185 to date a 6× microscope has been used with constant irrigation and suction as described by Shambaugh.

Starting with Case No. 222 the size of the fenestra has been measured and noted. It varies between 4.5 and 2.25 mm. in length by 1.0 - 1.25 mm. in width; the average size being 3.25 × 1 mm.

In all cases, the fenestra has been created with a polishing burr. The edges have been cleaned with dental picks and a gold burnishing "burr" used to eburnate the bone. Utmost care has been exercised in obtaining complete hemostasis and in insuring against bone dust in the fenestra.

SELECTION OF CASES AND TESTS FOR CLOSURE.

The present analysis is based on 170 cases (through Case No. 314) operated (without stopple) before July 1, 1949. Of these, 153 were operated since October 22, 1946, when the last stopple operation was performed. They are, therefore, consecutive cases except that we have been forced to omit 15 cases on whom we have been unable to perform the necessary follow-up tests.

The routine tests of hearing included "loss for speech" measured by P.A.L. Auditory Test No. 9 (spondees), "average loss for the speech range" (measured as the average pure tone loss at frequencies 512, 1024, 2048 and 2896 cycles per second), and the fistula test for patency of the fenestra in doubtful cases. The tests of hearing have been fully described elsewhere.[3,4]

The first complete post-operative tests were usually done during the second month after operation. The plan of follow-up called for another complete test at six months, one at a year, and then at annual intervals thereafter. For practical reasons this schedule could not be followed very closely. Some patients were tested more frequently, particularly when they complained that they were losing their hearing again. Actually, for the group as a whole, the tests were scattered nearly at random except for a gradual decrease in frequency over the first year and a half. The annual testing thereafter has been a little more regular.

In the 170 fenestration operations there were 20 that we have classed as failures because they did not show as much as 10 db gain for speech at the first complete post-operative test. The causes of these "immediate failures" have already been analyzed[1] but it is worth recalling that the incidence of such "failure," reckoned in the basis of all (non-stopple) original fenestrations including those not followed up, is 20/185 — 10.81 per cent. Because many (13) "failures" did not return for follow-up, we have omitted from our calculations all "failure" because of very early closure. In other words, we reckon the incidence of closure on the basis of the "initially successful" operations that gave at least 10 db gain for speech.

CLOSURE.

Closure of the fenestra is suspected when any follow-up test shows that the patient's threshold for speech is 10 db or more higher than it was at the first post-operative test. Closure is proved by a negative fistula test, *i.e.* no nystagmus or vertigo when gentle pressure is applied to the skin graft over the fenestra. Furthermore, the diagnosis of closure was confirmed at operation in the 14 cases on whom "revision" was attempted. One case (No. 5) showed a dense fibrous scar in the fenestra. All of the 13 others were true bony closures.

Only the cases proved closed by the fistula test (when it was clear) are counted in the calculations below, although a few others suffered more than 10 db loss of their initial gains for other reasons.[1]

Two cases are complicated, one by injury from an explosion just prior to "closure" and the second by some nerve deafness and rather serious psychological difficulties. In these two the evidence for closure is doubtful or incomplete. We arbitrarily include the former (No. 270) but reject the latter. This probably gives the fairest overall figure for incidence of closure.

We have in all, then, 21 definite and one presumed closures. This is 14.6 per cent of the 151 initially successful operations (without stopple) on whom we have adequate follow-up data. The percentage may still increase slightly as 15 cases are still in the second six month period after the operation when closure is still fairly frequent.

When closure occurred it occurred within the first year and often within the first six months after operation. We have tests on 73 cases after 1½ years or more (9 of them 3½ years or more) and not one has closed later than 12½ months after operation (see Figure 1).

The median of the intervals at which the 22 closures were last proved to be still open is 2.7 months. The median of the intervals at which they were first found to be closed was 7.5 months. The earliest closure was within 1 month. Only one case that is known to have been open at 9 months subsequently closed. The most probable time of closure, if it is to occur, is, therefore, during the fifth month after operation. Figure 1 shows that this figure has not altered significantly since the beginning of our series. Furthermore the incidence of closure seems to be constant. Our three successive groups of 50 cases each contain 8, 7 and 7 closures respectively.

The 132 successful cases that have remained open show a slight mean loss in hearing for speech (based on Test No. 9) since the first post-operative test: —2.77 db ± 4.7 db. The distribution of the losses about the mean is not skewed. This

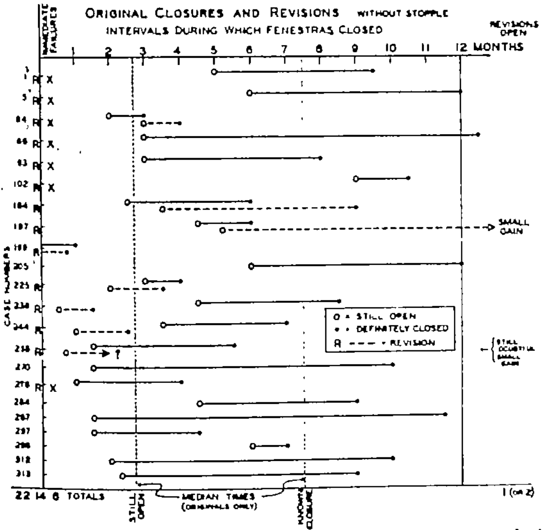

Fig. 1. The horizontal lines show the period after operation during which the closure took place. Each open circle indicates the last test at which hearing was still up to or within 10 db of the immediate post-operative level. The filled circle indicates the date of the first test at which there was clear evidence that closure had occurred. All intermediate tests in which the evidence was equivocal are omitted.

slight but significant loss of sensitivity does not seem to increase after the first year and apparently not after the first six months, but more cases are needed to confirm these latter impressions. This slight late loss of nearly 3 db should be added to the expected minimum residual loss of 20 db that we discussed in a previous paper.[1] The late loss may be due to a stiffening of the skin graft and other tissues as scar tissue becomes mature. Further details concerning changes in auditory sensitivity during the first year after fenestration are reserved for later publication.

CLOSURE IN RELATION TO AGE.

Closure is obviously due to a normal healing process. It seems to be the general opinion that young patients (in their teens) are less suitable for fenestration because their repair of broken bones is known to be more rapid and perhaps more effective than repair in adults. It is assumed, therefore, that closure of the fenestra is more probable in such patients.

Inasmuch as our series contains a number of patients under twenty years of age at the time of operation we have examined our data for possible correlations between age and the incidence of closure.[*]

First we calculated the mean age of the 23 patients whose original fenestras closed (31.0 ± 11.9 years) and compared it with the mean age of 170[†] other patients whose fenestras were known to be open (33.6 ± 9.0 years). The t-test shows that this small difference is quite insignificant and would be expected once in three times because of the large standard deviations.

As a more delicate test we grouped the patients by decades and compared the incidence of closure in each group. Four of our closures are in the 10-19 year group, giving an incidence of 33.3 per cent. On the other hand we have only 5 in the 30-39 year group, a percentage of 7.25 per cent. In the entire group from 20 to 69 years we have 19 cases or 10.5 per cent. Further calculations show that the difference between the 10-19 and the 30-39 year groups is significant at about the .02 level. The difference between the 10-19 year and the 20-69 year groups is also significant (at about the .04 level). All of the other differences would be expected to occur by chance oftener than once in twenty times. The data and calculations thus support the general impression that a fenestra in a teen-age patient is more likely to close than one in an adult. Because of the small numbers of cases involved we do not regard our data on this point as definitive, but they are certainly very suggestive.

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[*] The calculations below were made in April, 1950, before the final classification of several doubtful cases had been settled. On the basis of more recent data two cases (aged 28 and 31) were omitted from and one case (age 39) was included in the final series discussed in the previous section as compared with the group here analyzed for the effect of age. These shifts do not significantly affect the results to be presented.

[†] The series is larger than the series analyzed in the previous section as cases through No. 232 and some additional follow-ups on earlier cases were available.

A very important detail of the relation of closure to age, however, is found among the very youngest members of the series. *The fenestras of the three youngest, aged 10, 12 and 13 years, all closed.* The next six, aged 14 to 18 years inclusive, all remained open. In fact, the high incidence of closure among the teen-agers is due entirely to the three youngest. Otherwise, except possibly for the rather low incidence in the third decade, the closures seem to be distributed quite at random.

Our practical conclusion is, therefore, that the fenestration operation should not be performed before the age of fifteen years. Patients younger than this should wear hearing aids, in spite of the social advantage that would follow from a successful fenestration. This point becomes doubly important when we consider the very unfavorable outcome of attempts to "revise" closures, as described in the following section.

REVISION.

Fourteen of the 22 fenestras that closed were revised. Unfortunately revision was uniformly unsuccessful,[*] either because of immediate failure (particularly in the cases revised in 1946) or because, with one or possibly two exceptions, the fenestra closed a second time (see Figure 1). Only these 2 out of the 14 revisions can be considered even partially successful, with gains of 8 db and 14 db (equivocal, based on contradictory data) respectively. In addition one other case that closed has nevertheless improved by 6 db. All of the 11 others were made worse by the revision. Six ears were totally deafened at the operation, most of them by tearing the membranous labyrinth that had adhered to the new bone. The failures are easy to understand. The scar tissue makes the

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[*] In addition to the series here presented, one of us (T. E. W.) has also revised 81 fenestrations in which he employed the stopple cartilage. Adopting the same criterion of success, i.e. a gain for speech of 10 db or more, and classing as doubtful those on whom the data is insufficient or equivocal, the results are a little better than in the present series, i.e.

Successful:	28 per cent
Failure, including closures.	65 per cent
Doubtful:	7 per cent

If the final result is compared to the status before the original operation the percentage of "successful" cases becomes 38 per cent. The percentage of original closures was far higher with the stopple than in the present series since the stopple was abandoned.

operation far more difficult surgically, and five of the immediate failures are among our first six revisions.

The extraordinarily high percentage of closures of the revised fenestras raises a question as to whether the second closure is due in some way to the previous operation. Does the old scar tissue favor closure? Have the tissues been "stimulated" in some way to more active repair? Does the original closure automatically select the cases with the strongest constitutional tendency to form new bone and close the fenestra? Against the idea that the previous operation favors closure is the rather scanty data in Figure 1. Here it is clear that on the average each ear closed the second time after about the same length of time that it did the first time. Even if closure has been made more likely it has not been made much more rapid. The "constitutional" theory will be tested when we have accumulated enough cases in which the opposite ear is fenestrated. This now seems to be the operation of choice in case of "failure." In any case we have abandoned the classical "revision operation" because of the very unfavorable results here reported.

SUMMARY.

Follow-up tests of hearing and for closure of the fenestra have been conducted over periods from 6 months to 5 years on 170 patients.

Closure of the fenestra occurred in 22 cases or 14.6 per cent of the 151 initially successful cases. Closure occurred in all three of our patients who were less than 15 years old, but there is only a slight relation if any to age from 15 years onward.

Closure usually occurred between 2.7 and 7.5 months after operation. Only one of the 22 closed after more than 9 months and none after one year.

The cases that did not close show a mean loss of hearing for speech of 2.77 ± 4.7 db that occurred during the first year after operation but (on the average) did not increase further.

The operation was "revised" in 14 cases of closure. The results were very unsatisfactory. Six cases were immediate failures and six more closed again after about the same interval as after the original operation. Only one revised fenestra is apparently open after one year and the gain is only 8 db. We have abandoned the revision operation. There has been no apparent relation between closure and technique.

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INTRALARYNGEAL CORRECTION OF BILATERAL ABDUCTOR PARALYSIS: A MODIFICATION OF THE THORNELL OPERATION.*†

HANS VON LEDEN, M.D.,
Chicago, Ill.

The increase of goiter surgery in the past 50 years has caused a corresponding rise in the number of vocal cord paralyses. Trauma of the recurrent laryngeal nerves at the time of surgery or during the postoperative period has produced numerous instances of bilateral abductor paralysis with its crippling and depressing effects on the unfortunate victims; yet it has been only during the past decade that methods have been devised for the successful correction of this dread complication.

The ideal solution of the problem would, of course, be the restoration of function for the paralyzed posterior cricoarytenoid muscles and a return to the normal physiology of the larynx; however, all attempts to reach this goal, whether by nerve anastomosis or by muscle transposition, have ended in failure. Other efforts have been directed toward widening of the glottic opening to establish an adequate airway without sacrificing the voice of the patient.

Early attempts to achieve these results by intralaryngeal cordectomy or ventriculocordectomy failed, due to the development of postoperative stenoses of the larynx. An arytenoidectomy was first performed by veterinary surgeons for relief of dyspnea in roarer horses, due to recurrent nerve

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*Submitted as Candidate's Thesis to the Chicago Laryngological and Otological Society in March, 1950, and read in abstract at a meeting of the Society on April 3, 1950.

†From the Departments of Otolaryngology, Stritch School of Medicine, Loyola University, and St. Francis Hospital, Evanston, Ill.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Aug. 7, 1950.

paralysis. A thorough survey of the literature reveals one successful case of arytenoidectomy by laryngofissure reported by Baker[1] in 1916; however, his work was passed over, and, in 1932, Hoover[2] followed the example of Killian, Dahmann and Albrecht by performing several satisfactory submucous resections of one vocal cord. Other surgeons failed to duplicate these results and, in 1936, Loré[3] suggested a more extensive submucous resection of one vocal cord with complete removal of the arytenoid cartilage. Unfortunately, all of those operations resulted in partial or complete loss of the voice.

In 1939, King[4,5] described his famous operation for the surgical treatment of bilateral abductor paralysis and stimulated new interest in this field. Originally he conceived the idea of transposing the anterior belly of the omohyoid muscle to the arytenoid cartilage, but he[6] soon discovered that his best results were obtained in those cases in which the arytenoid cartilage was disarticulated and fixed in lateral displacement together with the corresponding vocal cord. His success in widening the airway while preserving the voice encouraged many others to follow his example, and his technique has been employed with good results by Morrison[7] and others.

In the following year, 1940, Kelly[8,9] described a procedure, in which arytenoidectomy was carried out through a small window in the thyroid cartilage. Great interest greeted this new method, and several modifications were introduced in an attempt to perfect it. Galloway[10] suggested mobilization of the arytenoid cartilage and its fixation to the extralaryngeal structures outside the thyroid window. A similar method of transfixing the arytenoid cartilage was described by McCall and Gardiner.[11] Wright[12] sutured the extreme posterior end of the vocal cord to the perichondrium on the outer surface of the thyroid cartilage. Orton[13,14] used Trotter's[15] lateral transthyroid approach, and Woodman[16] removed a portion of the thyroid ala to obtain better exposure.

While all of these techniques were successful in a large number of cases, Kelly[17] himself was the first to admit the difficulties encountered in the extralaryngeal approach. In an excellent summary written in 1944, he calls attention to the difficulty in exposure, to the elusion of the arytenoid cartilage, to the breaking of sutures on coughing or vomiting, to possible lacerations through the laryngeal mucosa, and to the breakdown of cartilage in elderly patients, with subsequent delay in healing.

Two years ago, in an effort to overcome those difficulties, William Thornell,[18] of Cincinnati, described a new intralaryngeal approach to the surgical correction of bilateral vocal cord paralysis. The Thornell operation is founded on the use of the Lynch suspension laryngoscope and consists of complete intralaryngeal arytenoidectomy with lateral fixation of the corresponding vocal cord by electrocautery.

Since this first report 22 such operations have been successfully carried out, and several modifications have been introduced. In each case, the patient has been observed for a period of several months after surgery to permit an accurate appraisal of the functional result. Thornell[19-21] has performed seven of those operations and has added the use of an O'Dwyer intubation tube and an acrylic obturator. Havens[22] and his colleagues at the Mayo Clinic have performed 11 successful intralaryngeal arytenoidectomies and are very pleased about their results. Edward King,[23] of Los Angeles, has used this procedure twice, and I am reporting two cases that I operated upon last year. These two patients had developed bilateral vocal cord paralysis as a result of thyroid surgery. In both cases I employed a modification of the original Thornell operation, and obtained excellent results with this technique.

OPERATIVE TECHNIQUE.

In place of the cumbersome suspension apparatus, Seiffert's self-retaining laryngoscope (see Fig. 1) was used for exposure of the operative field; the head of the patient was supported on a mechanical head rest. Preoperative medication consisted of nembutal (1.5 gr.) at bedtime and morphine (one-eighth gr.) and scopolamine (1/200 gr.) one hour before surgery. Intravenous injection of thiopental sodium (U.S.P.)

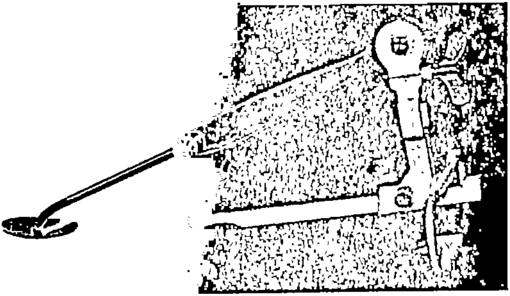

Fig. 1. Seiffert's self-retaining laryngoscope.

was supplemented by inhalation anesthesia of nitrous oxide and oxygen through the tracheotomy cannula. The operative area was further anesthetized by the local application of 2 per cent pontocaine.

After abolition of the pharyngeal reflexes and complete relaxation of the cervical muscles had been obtained, Seiffert's self-retaining laryngoscope was introduced and the larynx was exposed. An incision, 1 cm. in length, was then made over the superior surface of the arytenoid cartilage and extended anterolaterally into the aryepiglottic fold, as described by Thornell (see Fig. 2). The wound edges were separated, and the arytenoid cartilage was identified. It was stripped of its various muscular attachments by sharp submucous dissection, and disarticulated at the cricoarytenoid joint. In addition to the Lynch operating instruments, two very sharp knives, a straight bistoury and an angular bistoury with double edge blades (see Fig. 3), were adapted by me for this part of the procedure, because it is extremely important to avoid injury of the mucous membrane on the lateral wall of the larynx.

Following complete removal of the arytenoid cartilage a guarded curved electrocautery tip was inserted deeply through the incision along the course of the thyroarytenoid muscle, beneath the vocal cord and into the space previously occupied

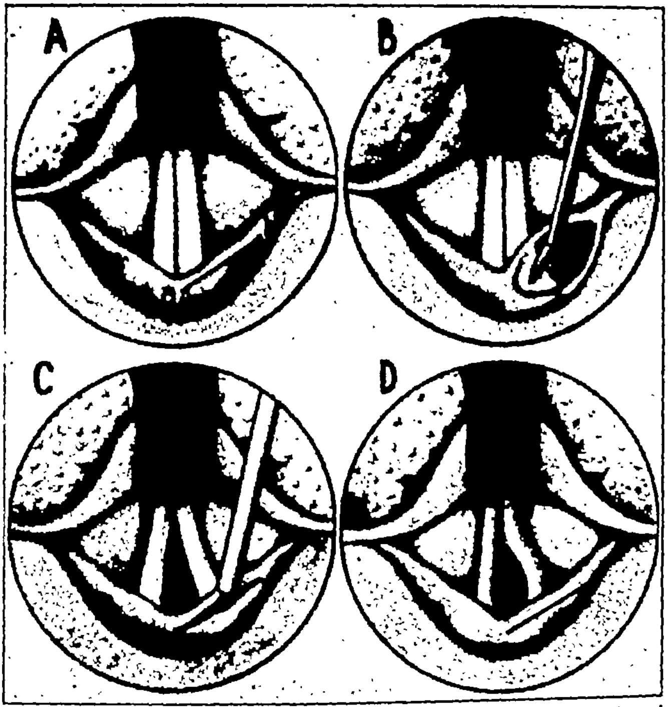

Fig. 2. Schema showing (A) the incision (a-b) over the arytenoid area extending into the aryepiglottic fold; (B) position of the arytenoid cartilage, tightly held by a grasping forceps; (C) lateral displacement resulting from the removal of the arytenoid cartilage, with the guarded electrocautery point in position for cauterization; (D) lateral displacement of the posterior two-thirds of the vocal cord. (Reproduced from the Archives of Otolaryngology, by courtesy of Dr. William C. Thornell.)

by the arytenoid cartilage. Further lateral fixation of the posterior half of the vocal cord is thus obtained by the resulting contraction from the electrocautery. A small amount of adrenalin (1 to 10,000 solution) was used for local infiltration in one of the cases, but bleeding was minimal in both operations and readily controlled with the electrocautery unit. The incision was allowed to remain open to afford drainage.

An acrylic obturator was then inserted between the cords (see Fig. 4); it was anchored inferiorly to the flange of the tracheal cannula and superiorly to the skin of the cheek. This obturator is tolerated well and causes surprisingly little reaction to the soft tissues of the larynx. I feel that its use is particularly important in cases where a previous laryngeal

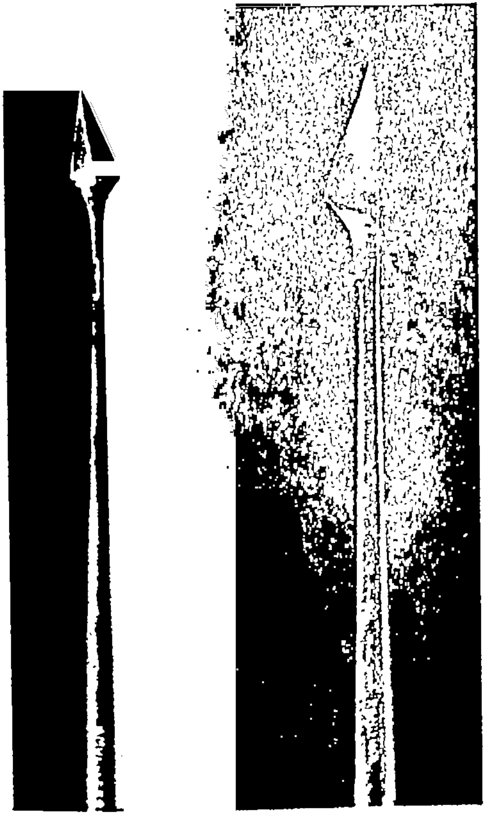

Fig. 3. Straight and angular bistoury knives mounted on long laryngeal handles.

procedure has been carried out with subsequent scarring of the laryngeal structures. One of my cases falls within this group; it is, I believe, the only case on record in which a successful intralaryngeal arytenoidectomy has been performed on the same side as an unsuccessful extralaryngeal operation.

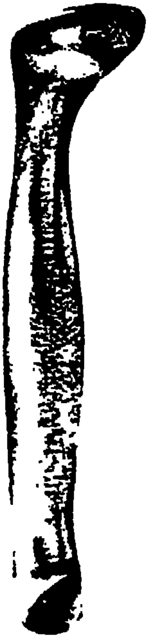

Fig. 4. Acrylic obturator.

CASE REPORTS.

Case 1: The patient, a 48-year-old white woman, was seen at St. Francis Hospital on Feb. 26, 1949. She presented a history of four thyroidectomies over a period of 28 years. Following the last operation in May 1948, she had noticed hoarseness, and in October 1948, respiratory difficulties developed. Her symptoms had become progressively worse since that time, forcing her to spend most of the time in bed.

At the time of my first examination, the patient was sitting up in bed, in acute respiratory distress, with marked dyspnea and a very loud laryngeal stridor; she was very nervous and jittery. The inspiratory crow was so noisy that it could be heard at the other end of the hall, in spite of closed doors. Indirect laryngoscopy revealed clockwise rotation of the entire larynx, with fixation of both vocal cords in the midline. The remainder of the ear, nose and throat examination revealed extensive scarring of the soft tissues of the neck.

An emergency tracheotomy was carried out, followed by immediate relief in breathing. During the convalescent period the patient developed a lobar pneumonia, an acute otitis media, and right heart failure, and her release from the hospital was delayed until March 17. Seven weeks later, on May 5, she was again admitted to the hospital complaining of nervousness, shortness of breath, pain in the left chest, and swelling of the lower extremities of four days' duration. The diagnosis on this admission was hypothyroidism and hypoparathyroidism with cardiac decompensation, and secondary bronchopneumonia. Under indicated therapy she improved very slowly until her discharge from the hospital on Aug. 13. She continued under strict medical observation, until her general condition had returned to normal and she was pronounced ready for intralaryngeal surgery.

On Dec. 18, 1949, the patient re-entered St. Francis Hospital, and on the following day a left arytenoidectomy was carried out through Seiffert's self-retaining laryngoscope, as outlined above. The left side was chosen for this procedure, because vibratory excursions of the right cord were more pronounced, although the rotation of the larynx made an operation on this side technically more difficult.

The postoperative course was uneventful, except for slight fever and increased pulse rate during the first three days. The acrylic obturator was removed on the fourth day. The moderate postoperative edema subsided quickly. The patient was discharged on the eighth postoperative day. She was again hospitalized on Jan. 24, 1950, when the tracheotomy stoma was closed under local anesthesia.

Since the operation the patient has lost her nervousness and all difficulties in breathing. She is taking care of her home and fulfills all the duties of housewife, though she has been advised by her cardiologist to avoid unnecessary strain. Her voice is clearer than it was at my first examination, although somewhat weaker.

Case 2: The patient, a 52-year-old white woman, was first seen in my office on May 31, 1949. She had had a thyroidectomy in July, 1948. Three months later she had noticed slight difficulty in breathing; this had become progressively more severe, until she had been forced to give up her position as a secretary and refrain from any and all exercise. She also complained of severe nervousness, loss of 40 pounds in weight, and stiffness of the neck, apparently due to muscular spasm from keeping the head tilted backwards in the position of best respiration.

On laryngeal examination, both vocal cords were found to be fixed in the midline, with only a 1 mm. glottic opening present. A low transverse cervical-thyroidectomy scar was noted. The patient was thin, extremely nervous and very worried. She had an inspiratory stridor, which became pronounced with slight exercise or emotional changes. A general medical examination by an internist revealed hypertensive heart disease and a severe anxiety state. Under indicated therapy her general condition improved, without any change in the laryngeal findings.

In October 1949, a King operation was carried out on the left side. During this operation the arytenoid cartilage broke in two, and the vocal process was anchored to the thyroid ala. At the close of the operation the glottic rima was about 5 mm. wide, but subsequent examinations indicated a narrowing of the airway to about 2 mm., probably the result of a broken suture. As a result, the patient was able to close her tracheotomy cannula at rest and during mild exercise, but was unable to return to her normal activities. She had gained 10 pounds and had lost some of her nervousness.

On Nov. 28 the patient again entered St. Francis Hospital, and a complete arytenoidectomy was performed through Seiffert's self-retaining laryngoscope, as described above. In order to preserve the vibrations of the intact right cord, it was decided to operate again on the left side, which had already been subjected to the above mentioned operation by the extralaryngeal approach. At this time the remaining portion of the arytenoid cartilage was removed by submucous resection. The scar tissue resulting from the previous operation was divided to permit further lateral motion of the left cord. The curved electrocautery tip was then inserted repeatedly along the posterior one-half of the left vocal cord, and the desired lateral fixation was obtained. Great care was taken in inserting the acrylic obturator into the newly formed triagular opening at the posterior end of the larynx.

The postoperative course of this patient was entirely uneventful. The small amount of edema began to subside on the third postoperative day, and the patient was permitted to get up on the same day. The acrylic obturator was left in position for a total of five days. The patient was dismissed from the hospital on the tenth postoperative day. She was again hospitalized on Jan. 24, 1950, when the tracheotomy stoma was closed under local anesthesia.

At the present time, the resulting glottic opening, as viewed indirectly, is 4 mm. wide posteriorly. Shortness of breath on exertion and the inspiratory stridor have completely disappeared. The patient is able to lead a normal existence, and has returned to her former position as a secretary. Her voice showed little change after the arytenoidectomy; it is hoarse, but adequate for all purposes, including prolonged telephone conversations. She has had a further 10 pound gain in weight and has lost her anxiety.

CONCLUSION.

I have found the intralaryngeal approach for surgical correction of bilateral abductor paralysis most satisfactory. I fully agree with Thornell[21] that "arytenoidectomy from within the larynx has been found feasible and not particularly difficult for one trained in the use of the suspension apparatus." Modification of the Thornell operation by the adaptation of the self-retaining laryngoscope and additional dissecting instruments was intended to simplify the procedure and increase its popularity. It is my firm belief that laryngologists trained in the use of the suspension or self-retaining laryngoscope will prefer this intralaryngeal approach. "For those surgeons without such training and equipment, the King operation or one of its modifications will and should continue to be the operation of choice."

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KERATOSIS OF THE LARYNX. REPORT OF A CASE WITH UNDERLYING CARCINOMA IN SITU.

GEORGE R. GORDON, M.D.,
Birmingham, Ala.

In reviewing the literature on keratosis of the larynx, one finds surprisingly little has been written on this most important subject. Particularly is this true in the era before 1940. This is all the more surprising since this most important lesion carries with it the stigma of being precancerous. Fortunately in the past decade several excellent articles have been written by such authorities as Clerf, Graham, New, Erich and several others. The textbooks also pay scant attention to this condition and often dismiss the subject with one or two inadequate paragraphs. Adding to the difficulty is the fact that there appears to be a definite diversity of opinion among the laryngologists and pathologists as to the terminology to apply to the various hyperplastic changes of the epithelium found in the larynx. One often finds such terms as keratosis, hyperkeratosis, leucoplakia, pachydermia, laryngis, hyperkeratosis laryngis and others used synonymously by some, while others consider each a distinct and separate entity.

Clerf[1] states that keratosis, hyperkeratosis or leucoplakia is primarily a localized benign hyperplasia of the epithelium, which does not invade the submucosa. This epithelial hyperplasia of the larynx may assume varying gradations from that of a simple profuse thickening as seen in chronic inflammatory reactions to that of large papillomatous masses that may fill the entire larynx. The whitish surface, which appears smooth or granular or may exhibit elevations or villus projections, stands out in contrast to the surrounding cordal mucosa, which often is the seed of a chronic inflammatory process.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 28, 1950.

New and Erich[2] differentiate epithelial hyperplasia from keratosis or leucoplakia in that the former consists of masses of piled up epithelial cells in contrast to the latter, where one finds a hyperkeratosis of the epithelium with a much thickened mucous membrane with epithelial down growths.

Pachydermia as considered by Hayes Martin[3] is a counterpart of the leucoplakia found commonly in the oral mucous membrane. The differential diagnosis of pachydermia from that of the other keratoses of the larynx is difficult and often impossible. In essence, he states that it is a response of these tissues to long standing chronic irritation and for the development of this lesion, the nature of the irritant is of less importance than its chronicity.

The term hyperkeratosis seems to describe most accurately the lesions discussed in its various gradations by the different authors.

These keratoses are rarely associated with similar changes in the oral mucous membrane as were seen in the case to be described.

Hayes Martin states that the percentage of intraoral keratosis or leucoplakia is extremely low in leucoplakia of the larynx or in the association with carcinoma of the larynx. He goes so far as to state that rarely is a case of leucoplakia of the larynx associated with oral leucoplakia.

Some factors stand out in all cases of keratosis of the larynx: *1.* there is a local keratinization of the epithelium without invasion of the submucosa. In discussing keratosis in general, Reiman considers it to be primarily a condition in which there is shedding of the superficial layers, the cells piling up and producing islands known as keratinized islands because of the excess keratin in the cells. *2.* In all forms of keratosis appearing on the vocal cord, there is one change commonly observed which has elicited the widest interest and speculation: it is the variable amount of inflammatory reaction noticed in the submucosal layer. This has caused much comment by Graham, New, and Erich and others as to its prognostic importance. This inflammatory reaction is diffuse throughout the mucous membrane of the larynx and shows areas of intensity about the base of the individual lesion. Graham[4] remarks that the presence of this inflammatory reaction is of the utmost importance and that a long step forward in determining the underlying cause of the whole process will have been made when this phenomenon is explained.

ETIOLOGICAL FACTORS.

While the causes of keratosis appear still to be in the realm of theory, there are predisposing factors well established in the minds of most otolaryngologists. Outstanding among these is that of long standing irritation. This is in keeping with the observation that keratosis is frequently associated with laryngitis of long standing. Hayes Martin flatly states that keratosis of the upper respiratory tract is a response of the mucous membrane of this tract to long standing chronic irritation. He further evaluates this statement by saying that in the development of the lesion, the nature of the irritant is of less importance than its chronicity. The lesion is most commonly observed in male adults who are excessive smokers.

Virchow[8] noted its appearance in alcoholic addicts or excessive voice users such as street hawkers. On the other hand, Clerf does not feel that excessive use of the voice or alcoholism is important.

Much work has been done experimentally and clinically in regard to the disturbances of vitamin A metabolism as related to keratinization of the skin. Along this line, Wallbach and Howe[9] have reported changes in epithelial structures following the deprivation of fat soluble vitamin A in experimental studies on rats. In these experiments which have been borne out by clinical evidence, it is found that various parts of the respiratory tract will undergo a keratinization of the epithelial surface when vitamin A metabolism was disturbed. Graham[4] was able to report a most significant case of keratinization of the larynx in which a remarkable regression of the entire lesion was observed as soon as attention was given to the vitamin intake in the patient's diet; this, despite a histological diagnosis of carcinoma in association with keratosis.

PRECANCEROUS LESION.

Many writers have applied the term precancerous lesion to this type of pathological change seen in the larynx. Where one finds decided hyperplasia of the epithelium of the larynx in response to long standing chronic inflammatory reaction, this hyperplasia frequently suggests early malignant changes. Crawford, Negus[6,7] and others have repeatedly remarked that localized or diffuse overproduction of the superficial epithelium offers considerable difficulty in deciding whether malignant changes are commencing in deeper layers of what is potentially a precancerous lesion. It is impossible in examination of such tissue to state categorically which ones will develop cancer and in which recovery will occur.

Morrison[8] reported a case of keratosis in which a carcinoma apparently developed as a single small island of malignant cells in the subepithelial layer beneath a mucus gland. New and Erich[2] have reported 10 cases, two of which developed a squamous cell carcinoma. In both cases the carcinoma was discovered only histologically. Clerf cites four cases of keratosis observed over a period varying from two to four years, which eventually developed carcinoma.

Coplin[9] appropriately stated that histology rarely, if ever, tells us what a cell is going to do before it does it, although many of us are deluded into the belief that we can deduce the future of a cell from a study of the past, present or both. Most authorities having concluded that chronic inflammatory changes in various forms often precede the appearance of malignancy, that keratosis of the larynx reveals just such a change, and that in most instances it has been definitely shown to have undergone malignant degeneration; it should, therefore, be considered a precancerous lesion of the utmost importance and treated as such, preferably by surgical removal.

Jackson,[10] in his book, Diseases and Injuries of the Larynx, states that leucoplakia of the larynx is definitely a precancerous lesion. It is impossible clinically to determine whether such a lesion has undergone malignant change in its early stages, and it often requires considerable histological study. In the broader sense, however, such a lesion presents a great potential danger and should be treated by surgical removal.

PATHOLOGY.

Grossly, one may find a simple profuse thickening or localized areas of thickening of the epithelial, appearing in the form of single or multiple chalky, white elevations on the surface and edge of one or both vocal cords. Such localized areas of thickening may be considered as small masses of hyperplastic squamous epithelium, which at times may suggest papillomas but lacking the usual characteristic arrangements. The basement membrane is usually regular but may push into the submucosa in small irregular projections. In most instances a good basal layer of epithelium is observed. In the submucosal layers changes of an inflammatory nature are seen with localized areas of intensity about the base of the individual lesion. There are present many cells of an inflammatory origin with numerous thin walled vessels, fibrosis and edema. Those lesions which show considerable inflammatory changes and great irregularity of the basement membrane should be dealt with immediately in the same manner as early carcinoma despite the lack of clinical or histological evidence of the presence of carcinomatous changes.

SYMPTOMS.

While the laryngoscopic picture may vary greatly from that of a diffuse infiltration to that of localized areas of thickening or even papillomatous changes, there is commonly just one symptom, mainly, a change in voice, ranging from a slight huskiness to a coarse hoarseness. No pain, dyspnea or allied laryngeal symptoms are observed until very late in the course of the disease, when it has progressed into the stage of carcinoma.

TREATMENT.

There is no doubt that keratosis of the larynx frequently offers considerable difficulty in deciding whether or not malignant changes are already commencing in the deeper layers. Many otolaryngologists and pathologists declare it is impossible to determine that such a change has occurred other than by histological study, and even then a definite diagnosis is often difficult. This is aptly brought out by the two cases of Graham in which carcinoma was determined only through histological study. Coupled with this is the fact that one cannot prognosticate clinically or histologically whether any given case will or will not proceed into a condition of carcinoma. The consensus is that keratosis is a precancerous lesion and should be considered and treated as such. A progressive change in appearance or an increase in symptoms gives warning that a simple process, possibly present for years, may be changing its character. Certainly all cases warrant and demand histological study through biopsy even though this may not prove anything as demonstrated in Morrison's case and the case to be detailed here. In certain cases patients recover on a regimen of no smoking, vitamin therapy, vocal rest and removal of the keratotic material by various appropriate surgical measures. These measures are often inadequate and keratotic masses reappear despite frequent removal and even stripping of the cords as described by Putney, Clerf and Loré.[11,12]

Jackson and Jackson[10] state that the treatment of precancerous conditions when recognized is urgently called for. Keratosis requires removal, notwithstanding the fact that in many cases it may exist for years before developing malignant qualities. When the lesion's appearance is suggestive of carcinoma, then laryngofissure is definitely indicated, even though none may be found histologically. Close observation and repeated examination of all these lesions is imperative along with biopsy, so that one can detect the earliest possible changes in a lesion so predominantly precancerous and in which so much difficulty arises in our ability to determine even histologically if carcinoma is arising.

REPORT OF CASE.

J. W., male, white, age 36, first seen on Aug. 18, 1949. The reason for his visit was to arrange for a tonsillectomy while on vacation. During the course of history taking, it was noted that the patient spoke with some degree of hoarseness. On inquiry the patient stated that this symptom had been present in a varying degree from four to five years. He attributed this condition to his job in a foundry where he was subject to varying extremes of temperature and dampness. He smoked several packs of cigarettes daily and was a moderate drinker of alcohol.

Examination revealed this patient to have a rather diffuse leucoplakia of the buccal cavity on either side. Mirror examination of the larynx showed the right vocal cord in its middle third and extending slightly beyond this area toward the anterior commissure to be thickened, ragged, grayish-white and frayed. There seemed to be no distinct line of demarcation from the surrounding tissue. The remainder of the laryngeal mucosa and the left cord was entirely free of any suspicious change.

This patient was admitted to the hospital on Aug. 18, 1949, for direct laryngoscopy and biopsy.

General physical examination was essentially negative, laboratory studies within normal limits, Kahn negative.

Gross report of biopsy: "A piece of white, pink tissue measuring 0.25 by 0.2 cm., which is soft but not friable. Microscopic description: the tissue shows slight hyperkeratosis with parakeratosis. A thickened, stratified squamous epithelium and numerous cells of chronic inflammatory reaction in the subepithelial tissue. The epithelial cells are reasonably well arranged in regular fashion and there is no great variation in nuclear size. There are some mitotic figures seen. There is one area of stratified squamous epithelium where the cells are less regularly arranged and the demarcation between epithelium and connected tissue is not particularly sharp. One area shows apparently deeply situated epithelium, but this may well be merely the effect of a tangential cut. Base features suggest that this is a hyperkeratosis, but if there is the appearance of carcinoma on direct visualization of the region, I would suggest repeat biopsy."

The result of the biopsy and the type of lesion were discussed with the patient, and it was decided to proceed with a laryngofissure. This was done on Aug. 31, 1949. A tracheotomy was not performed.

Gross description of removed tissue: A mass of tissue measuring 2.5 by 1.5 by 0.5 cm. One surface is hemorrhagic, the other surface is yellow in color. There are firm nodular areas 0.5 cm. in diameter in this mass.

Microscopic description: It is noted that the stratified squamous epithelium is greatly thickened in areas with great tags of thin masses, both as to width and to depth, and seems to invade some of the tissue. There is much chronic inflammation also seen in the section. Several sections made in the tissue show that in one portion of this section the squamous epithelium sends large columns deep into the tissue, and in these particular column areas there appears to be an invasion about their bases. The nuclei of the cells here are enlarged, versicular-looking, and occasional mitotic figures can be seen.

Diagnosis: Hyperkeratosis, squamous cell carcinoma of the larynx, grade I.

CONCLUSIONS.

Keratosis of the larynx is not a common disease. In association with oral leucoplakia it is rare.

Keratosis, while a relatively benign lesion, occurring chiefly in male adults, particularly in excessive smokers, carries with it the stigma of being a precancerous lesion.

While its etiology is unknown, experimental and clinical evidence tends to show that it may be related to some metabolic or food deficiency disease.

It is impossible, either clinically or histologically, to predetermine whether any given case will become cancerous. It is also frequently impossible to discover any malignant change other than through thorough biopsy or complete removal and serosections of the entire lesion.

By reason of their precancerous status, these cases should be kept under close observation and supervision. Any question or doubt as to the presence of malignant changes warrants the removal of the lesion by surgery. The status of leucoplakia is such as to lead many authorities to advocate surgery immediately; the surgery of choice is laryngofissure.

A case of hyperkeratosis of the larynx involving one cord in association with oral leucoplakia and exhibiting the presence of carcinoma determined by histological study is presented.

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ADDITIONAL APPENDIX.

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NUTRITION AND THE EYE, EAR, NOSE AND THROAT.*

(With Excerpts from the Literature.)

ISAAC H. JONES, M.D., Los Angeles, Calif.;
HAROLD S. MUCKLESTON, M.D., Pomona, Calif.;
EUGENE R. LEWIS, M.D., Los Angeles, Calif.,
and GILBERT ROY OWEN, M.D., Los Angeles, Calif.

Diseases that afflict mankind seem to fall into two broad classifications — those from without and those from within — the infectious diseases from without and the degenerative diseases from within. Infections may attack swiftly; we often observe the cause and effect within weeks or even days. Degenerative disease may take a long time to develop. The lack of the needed nutrients in the cell shows no immediate effect; only after this lack has continued do its effects become evident — in caries of the teeth or degeneration of the structures around the teeth causing them to fall out, in chronic arthritis or degenerative changes in the heart and other organs. There is another contrast between infectious and degenerative diseases. Great advances have been made in the control of infections; but, in the civilized world degenerative diseases are on the increase. To be sure, nowadays people live longer than they used to; obviously the increase in degenerative disease is to that extent more apparent than real — entirely apart from any consideration of defective nutrition.

It seems that the laws of nutrition, when finally clarified, will lose much of their present complexity, as so well expressed by Harding:[1] Einstein has recently put forth his "Unified Field" theory which interlocks gravitation and electromagnetics in a single equation — aiming to explain more

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*To be presented at the Midwinter Convention of the Research Study Club, Los Angeles, Calif., January, 1951.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 1, 1950.

and more phenomena by the use of a single formula. In studying the phenomena of metabolism we have resorted to fractionation — dividing each problem into its tiniest parts. The method is good and we are acquiring tentative answers to a large number of research problems. Each solved problem is like an identified fragment of a jigsaw puzzle; the time is approaching when we can fit these together, and as we see the pattern emerge we can more readily see what the fragments begin to mean.

Early in our studies of Nutrition it seemed that the internist was the one best fitted to observe and study the effects of deficiencies; but gradually it became clear that the tissues most easily observed are the skin and the eye, ear, nose, mouth and throat.

It seems clear that degenerative disease is due to poor nourishment. This concept might be briefly expressed:—A lack of the right food produces pathologic change in all tissues. As to vitamins, the main lack in our national intake is B Complex. For thousands of years bread had been good food. Then millers began removing vitamins and minerals, leaving only impoverished flour. Grain products now furnish one third of our food — and ninety eight percent of flour is white. Refined sugar is also a carbohydrate from which vitamins and minerals have been extracted, and sugar accounts for actually one seventh of the total food calories of the United States. In general, foods which spoil are good and foods which do not spoil are bad.

From the ideal and also the practical standpoint it is discouraging to face the fact that civilization affords foods of poor quality. It is so different from the days when vegetables came direct from the garden, milk direct from the cow, meats direct from "the killing". In those days the boy took the grain to the mill, bringing home fresh-ground meal and flour. Today's predicament is the unavailability of such foods; the only possibility is to make the best of a bad situation.

In this vein, as to the impossible ideal, Lee[2] makes the following suggestions: Eat no white flour or refined sugar.

As sweets use only honey, brown sugar, maple sugar or refined saccharin; use *fresh-ground* wheat or other cereals exclusively, because some vitamins become oxidized in a few days. Freshness is the measure of wholesomeness. The very flavor of whole wheat changes day by day after it is ground up — like coffee; the finer the grinding, the faster its loss of vitamins. Many people are now finding the answer in a home grinder that can be obtained at low cost from department stores. Pasteurized milk is relatively valueless as a source of the calcium it is so highly recommended for, because pasteurization destroys the enzymes necessary for calcium assimilation; so rely on raw certified milk, if your local Medical Association makes it available. Citrus fruits are not dependable sources of "C" unless fresh. Oranges may be devoid of "C" if over three months old, although not differing in appearance from fresh fruit. Leafy vegetables may lose all their "C" in one week of storage, so the only dependable source is the roadside farmer or your own garden. Meat is kept in cold storage until it contains only a vestige of its original vitamin content; fresh fowl and fish are preferable for vitamin content. Peanut products, fresh roasted nuts or peanut butter, are good high protein substitutes for meat. Potatoes, like wheat, can support life indefinitely if eaten baked or boiled, but not mashed. Mashing exposes "C" to air causing its destruction. Potatoes have about one third the "C" content of oranges. Bananas contain every essential nutrient. So, we can list as basic dependable foods: milk, wheat, potatoes, bananas. Each will support human life if not devitalized by tampering. Meat will stand in this category, especially if fresh.

As long ago as 1906 Ladd and Stallings[3] showed that the only excuse for using bleach is that it makes flour whiter. Bleaching also makes inferior flour look as "good" as superior flour. Bleach injures gluten. Even then the question was raised whether the U.S. Government should not prohibit the bleaching process because of its degradation of the bakery products. Their conclusions, even in 1906, condemned the process of bleaching. Lee[4] comments that bleached flour was declared in 1919 by the U.S. Supreme Court to be a threat to public health; however, the bleaching still continues. As to sugar — most people enjoy dessert; but Harding[a] points out that sweets make one feel well fed without being well fed.

At a recent Midwinter Convention of the Research Study Club we presented a simple method of finding out what a patient eats. This was printed and distributed among many — and adopted by some. It has been suggested that this be reproduced here.

* * * *

The nurse gives the patient a piece of paper on which there is an outline of "morning, noon and night" for two weeks. She then explains that immediately after each meal (while it is all fresh in mind) the patient is to write down what he ate; anything taken between meals is also to be written down. The patient reads his own handwriting to the doctor—at the end of two weeks. The doctor writes the word GOOD on one side of the page and BAD on the other side. He listens to the patient and interrupts as needed so that he can make notes under the heading GOOD or the heading BAD.

Analysis: You will feel a bit awkward for a while. Everyone does. However, you will be doing a great service to your patient if you simply have him cut down on everything that is made from white flour and sugar. As it were, they reduce the "high octane" of the fuel on which we travel. It is perfectly possible for our automobile to go along on a wretched gasoline — that is the trouble. We think that because we are getting along, everything is all right. Far from it. For health, human fuel requires a high-octane rating.

All of us are confronted daily with problems due, wholly or in part, to lack of the right foods. For example — a "stuffy" nose; non-suppurative sinuitis; lesions of the conjunctiva and cornea, angles of the mouth and tongue; beginning nerve deafness with no apparent cause; and "catching colds" frequently.

Before we used our present routine we wasted time collecting the information. Now we find that the whole procedure can be done in twenty or thirty minutes, including instructions to the patient. The patient reads his own handwriting while you make notes. Then you give a piece of paper to the patient — on the one side the word BAD and on the other side the word GOOD; the patient then puts down your instructions in his own handwriting so that he also will have no difficulty in reading it.

Almost invariably you will find that there is a lack in the B Complex. Liver is prescribed twice weekly: oil is brought to a very high temperature in the skillet, the liver is dropped in until it is crisp on the outside — then turned over. The result is what we call "Eskimo Pie Liver" — crisp on the outside and rare on the inside. Wheat germ is helpful — a tablespoonful every morning or every other morning, given with cereal. Of course whole wheat bread, if you can get it. Large doses of the B Complex are often helpful; the amount may be reduced after the patient returns for further directions.

As to the sugar, exhort the patient to use as little as possible from now on. He may look shocked — until you explain that you have something far better—refined saccharin—five hundred times sweeter than sugar. It is perfect; no bitter taste. Naturally, saccharin cannot be dropped on any cereal, but instead is put in the milk or cream.

Well, so much for a beginning! Having once started you will then enlarge your information and be better and better able to answer specific questions — "Is this good?" — "Is that bad?" If this routine is followed the results will be fair, good or excellent. By prescribing the right food we are truly physicians of the eye, ear, nose and throat. If we do not, we are neglecting an important service to the patient.

* * * *

One disease concerning which our knowledge is by no means commensurate with our interest and concern, is poliomyelitis. The rhinologist has been concerned with this disease for a long time. It was first thought that the matter was quite simple — infection enters through the nose and attacks the central nervous system. Forty-one years ago a virus was found in the degenerated areas of the nervous tissues; later it was thought that the cribriform plate was the portal of entry, and the zinc sulphate treatment came into vogue. The idea was to "toughen the mucosa" by spraying the zinc into the upper reaches of the nose. Although this treatment came to be used widely, it seemed so fantastic that we asked Dr. Leland Hunnicutt[6,7] to conduct experiments on dogs and monkeys for the Research Study Club. At that time the residents, internes and nurses of our County Hospital were all set to take this treatment. The histologic study by Dr. Hunnicutt showed a complete loss of olfactory epithelium and degeneration of the neurones leading from the surface. Because of these findings this treatment was abandoned at the hospital — and soon abandoned by everyone.

The opinion of authorities is well expressed by Bower.[8] Based upon his experience with 6000 cases in a four year period at the Los Angeles General Hospital Bower gives his up-to-date concepts of polio. The virus that causes this disease is the "Legio Debilitans". This virus occurs in three groups and representative strains from all three have been found. This disease of man has been reproduced by inoculating infectious human material into primates; and one strain has reproduced the disease in rodents. Whereas the virus may be recovered in only a low percentage of cases early in the disease by swabbing the nasopharynx, it may be obtained with relative ease and frequency for weeks and even months from stools — both of patients and of asymptomatic carriers. Bower states that in his hospital during this four year period doctors, nurses and attendants worked without masks yet none contracted the disease. (In contrast to this the 1934 epidemic had affected many nurses and several doctors in the same hospital.) During an epidemic people should be told to keep the hands clean constantly and to avoid excessive fatigue; no other steps have shown evidence of inhibiting the spread of the disease during an epidemic.

The National Foundation of Infantile Paralysis frankly states that we do not know how to prevent polio or how to stop it. The portal of entry is unknown. The usual methods of combating infection — serums, vaccines and chemotherapy — have had no effect. Quarantine has been abandoned as useless.

A recent experience illustrates our helplessness. A few days ago a Davis Cup champion aged 21 appears at 11 A.M. — and his match is to be at 1 P.M. His throat is so "sore" that he can't swallow. He had been unable to eat any breakfast. No fever, pulse 60, blood-pressure normal and a perfectly normal appearance in nose, nasopharynx and throat. He is told "there is no infection, just a rheumatic sore throat, in the muscles"; he is advised to drink water in large quantities; to wear a light sweater, and play tennis. The next morning he is comfortable, and the next day entirely well. At the same time another young man also aged 21 presented a similar picture to a colleague; but the mother was the anxious kind, so for that reason and that reason alone, the patient was put in the hospital — where he died in 48 hours of bulbar polio — confirmed by autopsy. How can we prevent such things? What sort of disease is this polio?

It is no wonder that many strange and diverse suggestions have been advanced about this mysterious disease — all of them based upon what we eat. One group[9] suspects alkaloid poisoning from the milk of cows which have been forced by the hot dry season to eat Jimson weed and cocklebur. Another group of reports has just appeared in a popular magazine.[10] Several suspect that the virus is not the cause but the product of the disease; others note that areas with high content of calcium and phosphorus have little polio — and recommend eating bone-meal; another remarks the resemblance of polio to shell-fish disease and suggests this poisoning as a cause. Perhaps the most plausible speculation, although it has not worked out, was that lack of the B Complex, especially "B_1", leads to pathologic change in central nervous tissues. The theory advanced by several clinicians[11,12,13,14,15] is that the symptoms of polio resemble those of beri-beri which is recognized as due to lack of the B Complex, especially "B_1"; that "B_1" and "B_2" are concerned with cell respiration — which is most important in nerve tissue; that epidemics of polio were unknown through the ages until one hundred years ago and that man's intake of "B_1" has been markedly reduced since the introduction about the same time of modern methods of milling wheat. These ideas are certainly of great interest and, at first thought, seem plausible. Local lesions due to the wrong food are observed in the eye, ear, nose and throat in patients otherwise apparently well and it seems reasonable that some such lesions could occur in the brain and cord. However, any nutritional disease like beri-beri and pellagra, continues on and on until the right food is given. In sharp contrast, polio often quits with the frost—as if cut off with a knife. Elaborate experiments with "B_1", by Elvehjem[16] and others[17] led to diametrically opposite findings: Mice deprived of "B_1" were actually *more* resistant to the polio virus. The outstanding point about polio is that it occurs in epidemics; this fact would seem to confirm it as a communicable disease.

* * * *

Looking backward, we come to realize more and more the value of the life work of Weston A. Price, D.D.S.[18] Three pioneers in Nutrition have been Dr. Price in the United States, Sir Robert McCarrison in India and the Cheshire Doctors in England. Dr. Price might well be called "The Charles Darwin of Nutrition." Turning from test tubes and microscopes to the hitherto unstudied evidence among human beings, Dr. Price sought the factors responsible for fine teeth among people who had them — the isolated primitives. The world became his laboratory. As he traveled, his findings led him to the belief that dental caries was merely one expression of physical degeneration from what he had suspected — nutritional deficiencies. Whole tribes and villages at varying latitudes, altitudes and temperatures provided many thousands of case studies which he carefully recorded by pen and camera. Specimens of the foods used by these isolated peoples were sent back to Cleveland, Ohio, for laboratory analyses—some of them over a period of years—to cover seasonal changes. Travelers, explorers and scientists have made it common knowledge that in their natural environment primitive peoples possess fine teeth. On this clue Dr. Price selected fourteen primitive groups, sufficiently isolated to have maintained themselves on their traditional diets. Many voyages carried him from villages in Switzerland and Gaelic villages in the Outer Hebrides to the Eskimos and Indians of North America, and to the Melanesian and Polynesian South Sea Islands. Thence he traveled to Africa, down to the Australian Aborigines, the New Zealand Maories, and back to the primitive Indians of South America in the high Andes and coastal plains. Wherever he went, Dr. Price found the same thing. Stalwart bodies were typical of primitives on their traditional diets — rich in essential food factors. Primitive peoples, no matter where located, who were successfully maintaining their tribal pattern from one generation to another were found to be living on highly nutritious foods. While tribal foods varied as to source and kind, each tribe had learned the foods essential to vitality and reproduction. Laboratory analysis of these foods showed them to be exceedingly high in proteins, vitamins, minerals and fat-soluble factors. In cranial and dental development contrasts are striking between primitive individuals and their modern counterparts living on "civilized" foods. Dr. Price noted changes in pattern — elongated facial structure, deformed dental arch and various other malformations. Well established racial patterns changed with the introduction of refined commercial foods; increasing impairments were observed in succeeding generations. The importance of nutrition during pregnancy has long been recognized, but Dr. Price's investigations showed that primitives understood and practiced preconception nutritional programs for both parents. Many tribes required a period of pre-marital nutrition and children were spaced to permit the mother to maintain full health and strength — thus fostering offspring of physical excellence. Foods of special value were rationed among the pregnant and lactating women, as well as the maturing boys and girls in preparation for future parenthood. These primitives, with their fine bodies, homogeneous reproduction, emotional stability and freedom from degenerative ills, stand forth in sharp contrast with those subsisting on the impoverished foods of civilization. This great contribution from a life's work has been available for many years — and yet its full meaning is only beginning to be appreciated.

Untimely death robbed us of the amazing Mickey.[19,20] He saw man as an integral part of the earth. The material part of man consists of a dollar's worth of chemicals which come from that thin layer of the earth's surface called the soil — except for the oxygen, nitrogen, hydrogen and carbon which come from its atmosphere. The soil upon which all life depends is a thin layer of an average depth of eight inches over the face of the land. The earth beneath is as dead and sterile as the moon. That thin film is all that stands between man and extinction. Not only that — deficient soil produces deficient men, animals and plants. When this mere film disappears man disappears—"and the place thereof shall know him no more". The Empire of Babylon was irrigated by the Tigris; the Tigris changed its course and Babylon became a desert. Vast stretches once forested and inhabited are now the Sahara.

In 1673 Archer[21] made statements which are pertinent today. He spoke of the great damage which comes upon most people by not knowing about their own "constitutions of body; in ignorance, they eat and drink simply to please the palate". "Many dig their graves with their teeth and cut off the thread of life sooner than is required by God or nature — besides living in sickness and disease". He reasons that each one should study his own food and thus be his own doctor. He sees the order of evolution in medicine — first healing; later prevention of illness; preservation of health comes first, restoration of health next. According to the old proverb that every man is a fool — or a physician — at forty, each should study "the friendly agreement or antipathy of food to his own body; and know the nature of all foods". Man is to be pitied who knows not the nature of what he eats. "Negligence in so necessary a knowledge has occasioned much sickness to many, and death to not a few".

From the time of Hippocrates until 1830, as Elvchjem[22] states, it was assumed that all foods supplied only a single universal "nutrient". It was in 1830 that Prout analyzed food into fats, carbohydrates and proteins; and it was not until 1906 that Gowland Hopkins called attention to a "special factor," to which Casimir Funk gave the name "vitamin" in 1911.

The otolaryngologist or the dentist has unusual opportunities to detect evidences of nutritional deficiency in the course of routine inspections. In emphasizing this, Mann[23] lists those most frequently found. Glossitis is perhaps the most important. Patients frequently complain of burning, soreness and ulcers of the tongue and inability to tolerate hot, spicy foods. Red, hypertrophied papillae at the tip and sides of a swollen "pie-crust" tongue suggest early pellagra. If the tongue is slick and "gelatinous" in appearance it suggests more chronic pellagra, a "B_2" deficiency or an anemia. Glossitis is a cardinal sign of danger although it does not tell what specific trouble is present. Almost always nutritional deficiencies coexist; it is rare to find only a single deficiency. The color and tissue tone of the mucous membrane is also an important indicator. An oral mucosa which is fiery red, but not swollen or easily bleeding, indicates early pellagra. In chronic pellagra the mucosa is dark red and pyorrhea is frequent. In scurvy the gums are dark red, spongy and bleed easily. Fissures at the angle of the mouth suggest "B_2" deficiency. Such lesions start as pale areas, usually bilateral and symmetrical. The angles are progressively eroded and macerated until a triangular, wet, "angry-looking" lesion results.

In the Birmingham Clinic 1102 infants and children with uncomplicated deficiency diseases were studied by Spies, Ewing and Mann.[24] The mothers of many of these children had deficiency diseases — more pronounced during pregnancy and lactation. It seems probable that some of the infants began to develop deficiency disease in utero. In these infants and children a period of poor general health preceded the appearance of lesions of pellagra, beri-beri or riboflavin deficiency. A study of 200 of these cases showed that characteristic lesions disappeared promptly on giving the appropriate synthetic vitamin. The promptness of response in many was startling. An example—12 months old boy; his mother "got down to skin and bones", diarrhoea and sore mouth during the last two months of pregnancy; had always preferred carbohydrate food; in the two years preceding this child's birth (the youngest of seven) the family had had no milk, family diet being corn meal, grits, dry beans and salt pork. This baby was breast-fed and, since mother's milk supply was scanty, when he was three months old she began giving him grits and cornbread soaked in water. A week before admission to hospital diarrhoea started; emaciated, dehydrated and acutely ill. Urine showed no nicotinic acid. First two days he received 125 mg. of niacinamide daily in divided doses. At the end of 24 hours diarrhoea had decreased; in 48 hours it had ceased, appetite returned and he received liberal foods suitable to his age; discharged on the fifth hospital day. Niacinamide was continued for a month; at home his food remained poor but he ate more than prior to this illness. He continued gaining for eight months when the weight leveled off. This is a sample of the experience in these 200 cases. It should be noted that the therapeutic effects did not manifest themselves so quickly in the mothers as in the children.

To determine the effects of heat-treated foods upon growth and development, Pottenger[25] conducted experiments for ten years, utilizing nine hundred cats of which records were completed on six hundred. His attention was directed to the need for such studies by the mortality among cats which had been fed cooked meats. These cats withstood operations poorly despite good surgical technic. In time, more cats were available than could be fed on scraps from the kitchen; so raw meat scraps were purchased — including muscle, bone and viscera. This raw meat was fed to a similar lot of cats, and within a very short time these survived surgery; they were healthier and their offspring more vigorous. This contrast was so striking that systematic feeding experiments were undertaken. Careful clinical notes were kept; in the case of death, autopsy was performed, cataloguing gross and microscopic findings; calcium and phosphorus determinations were made; and at the end of ten years all remaining animals were autopsied. In the first experiments one group received raw meats 2/3, raw milk 1/3 and cod liver oil; another group received cooked meats 2/3, raw milk 1/3 and cod liver oil. Those receiving raw meat reproduced normally, litters averaged five, lactation was normal, offspring were normal. Cats receiving cooked meat reproduced a heterogenous lot, each kitten differing from his litter-mates; abortion was common — about 25 per cent in the first generation up to 70 per cent in the second generation; dystocia and death in labor were frequent; kitten mortality was high, often due to failure of lactation; vermin and intestinal infestations were frequent. Of the cats on cooked meats. many showed "bones as soft as rubber". Kittens of the third generation all died within six months, thereby terminating the strain. Another group of cats, first fed on raw meat, were switched to cooked meat for six months and then returned to raw meat. Kittens from this group showed some stigmata although mothers appeared to be in good health. Subsequent generations improved as long as they received raw meat. Their resistance to disease, greatly diminished on cooked meat, gradually increased upon return to raw meat. Cats fed on raw meat maintained normal malar and orbital arches, nasal structures, dental arches and dentition. Adult cats on cooked meat began to show abnormal conditions in the mouth in from 3 to 6 months — gingivitis, paradentosis, loss of teeth — but no caries. Others fed on raw milk were compared with those fed on pasteurized, evaporated and sweetened condensed milk. Those on raw meat flourished; those on the altered milks fared badly. In brief, it appears that raw meat and milk are better for cats than altered meat and milk.

VITAMINS.

A review by Evans[20] of the recently established and still unknown components of the B_2 Complex describes two new factors, — Pteroylglutamic Acid (PGA) and B_{12}. PGA has already given promise of value in nutritional macrocytic anemia; and in conjunction with B_{12}, in sprue. Isolated only recently, B_{12} appears of value in Addisonian pernicious anemia. It is the most powerful therapeutic agent known; one millionth of a gram provokes hematologic response in man. Seven other members of the B_2 Complex have already been established.

Two patients had pernicious anemia, two had nutritional macrocytic anemia, one had non-tropical sprue. After intramuscular injections of B_{12} Spies, Lopez and Milanes[37] observed positive hematologic response in the two cases of pernicious anemia; also in the two cases of nutritional macrocytic anemia and in the case of non-tropical sprue. All cases showed clinical improvement, including well-being, alertness, general strength and vigor and relief of soreness and burning of mouth and tongue.

The plasma level of "C" and the prothrombin time were studied in one hundred and four cases of epistaxis by Neivert, Engelberg and Pirk.[38] Only eleven showed normal "C" and prothrombin time. The level of "C" was subnormal in 68; of these, 35 also showed subnormal prothrombin time. In 25 the "C" level was normal but the prothrombin time was subnormal. Vitamin therapy was applied in 62 of the 93 who were deficient in "C", in prothrombin time, or in both. The clinical response could be determined only in 36, of whom 27 were improved — suggesting that "C" as well as "K" has a place in the management of nasal hemorrhage.

Although hemorrhages due to prothrombin deficiency can usually be adequately controlled by "K", Butt, Magath and Seldon[39] present evidence that such benefit does not occur in patients with severe liver damage.

MINERALS.

It is too early to know what place isotopes may come to occupy in medicine. It is not too early, however, to be aware of difficulties and dangers which beset the way. Light upon this subject is shed by studies of plants, bringing out the remarkable advantages and also disadvantages. It is obvious that isotopes are new and powerful tools, permitting investigations not previously possible. Russell[40] shows how isotopes are used in studying plant growth — especially how plants absorb and utilize the nutrients obtained from soil. Isotopes act as "tracers"—a word which explains exactly their use in biologic research. They are used as indicators to show the behavior of normal elements; for example, if the biologist is working with radioactive phosphorus it is in order to investigate normal phosphorus. The Geiger counter is so sensitive that if one ounce of radioactive phosphorus were spread uniformly over the entire surface of the earth it would be possible to check whether the correct amount had been placed on every square inch. Isotopes can be detected by both the ear and the eye. Photographic plates are also sensitive and provide visual details as to the tracer's distribution. It is possible to measure the amount of nutrient absorbed in a few minutes by the tip of a root less than one-fourth inch long. The use of tracers is in its infancy; the first explorer's map of any region is never so precise as those of later observers. Careful thought must be given to possible damage. The isotope may affect the growth of the plant. To make matters worse, the plant may still appear healthy and yet be damaged. The isotope is a new tool, fascinating, of great potential value, but not without serious dangers.

With atomic energy man can create elements at will for use in medicine, as expressed by Lawrence.[31] Ordinary elements can be made radioactive — a good example is in the use of iodine in thyroid cancer. A new carbon has been produced — At.Wt.14 instead of 12; its course can be traced in the living body of plant, animal and man. We may learn why chlorophyll is the only agent in nature than can harness energy from the sun for creating the food on which we live. Carbon 14 may enable us to create food from simple substances, just as the plant does. Man, in fact, stands at the gateway of a new world — provided of course he does not commit suicide inadvertently.

In the light of recent advances Deuel[32] considers that the attitude toward the importance of fats should be changed. They are no longer to be considered as "optional", but rather as required food. In studies of the growth of rats it appeared that diets containing 20-30 per cent fat gave optimal results. It was also found that rats on high-fat intake could accomplish a greater amount of work than those on low-fat intake. The development of the isotope technic has made it possible to follow the various intermediates of fatty acid oxidation through their several pathways.

Calcium is required by animal and human bodies in larger percentage than any other element; as Albrecht[33] expresses it calcium heads the list of strictly soil-borne elements required in the nourishment of living tissues. As rocks are broken down to form soil there is an increase of active calcium. When heavy rainfall occurs and the temperature rises there is calcium depletion. Lower forms of life — like microbes, and higher forms — like plants and animals, all are part of this calcium picture. The distributions of the different plants and animals take their patterns very much according to the calcium supply.

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BOOK REVIEW.

Facial Paralysis. Being a Treatise on a Clinical Classification of Paralysis of the Facial Nerve. By J. Parkes Findlay, M.B., Ch.M., Honorary Assistant Aural Surgeon, Sydney Hospital. 47 pages with index and illustrations. Sydney, Australia: Angus & Robertson, 89 Castlereagh Street. 1950. Price 17/6.

This is a most interesting and useful monograph. The author has reviewed the anatomy of the facial nerve and its distributions. His comments on the history-taking and examination of the patient are very valuable.

The classification of facial paralysis which is offered is interesting: it is doubtful whether it will be quite generally accepted. One finds it difficult to agree, for instance, that the facial paralysis of sudden and complete onset is always accompanied by pain or, vice versa, that painless paralysis necessarily comes on gradually. It is difficult for this reviewer to agree that the differential diagnosis as presented by the author is entirely tenable. It is doubtful whether many will agree with the author in his dictum that "the treatment of vascular occlusion facial paralysis is surgical" and that "there is no clinical reason to delay the decompression of the nerve."

Whether the reader agrees with him or not, Dr. Findlay has presented a controversial subject in an interesting and provocative manner and the book is recommended to all interested in the facial nerve. T. E. W.

In Memoriam

SAMUEL J. KOPETZKY, M.D.,

1876-1950.

As we may be sure he would have wished it, Sam Kopetzky ried on his work with stimulating and tireless ardor up to day of his death, Nov. 13, 1950.

Born in New York City in 1876, Samuel J. Kopetzky gradud from the College of Physicians and Surgeons, Columbia iversity, in 1898 and later from the Royal University Clinic, rlin.

Sam was a true and fervent patriot; he served in the Army dical Corps in the Spanish-American War and both World rs, retiring with the rank of colonel. He was decorated h the Legion of Merit and the Silver Star.

Since 1939 he had been director of his department at the yclinic Hospital, professor of otolaryngology since 1920, on staff of the Manhattan Eye, Ear and Throat Hospital for ny years, consultant otologist at Beth Israel, Newark Beth ael and Vassar Brothers (Poughkeepsie, N. Y.) Hospital. his work on petrositis, France bestowed upon him the der of Chevalier of the Legion of Honor. The American

Laryngological, Rhinological and Otological Society, Inc., of which he was president (1937), bestowed upon him its gold medal.

Dr. Samuel J. Kopetzky was active in the work of all the medical societies to which he belonged, and he became president of the medical society of the County of New York and later of the medical society of the State of New York. At the time of his death he was a Fellow of the American Medical Association, the New York Academy of Medicine, the American College of Surgeons, the American College of Ophthalmology and Otolaryngology.

A facile and fluent contributor to the otological literature, Dr. Kopetzky was one of the founders and, from 1935 to 1940, was the editor of *Medical Week.* He was literary chairman of the *New York State Journal of Medicine.* At the time of his death he was president of the Metropolitan Chapter of the Association of Military Surgeons of the United States. Several textbooks bear his name.

Our memories of Sam Kopetzky will contain as his outstanding traits kindness, understanding, a stable though tolerant and fair judgment, a friendship unaffected by differences of opinion, a seeker after the truth, a soldier and a gentleman.

To his widow, Mrs. Anah Doob Kopetzky, his son, Karl A., his daughter, Mrs. Yvonne K. Sterling, his three sisters and his many friends, our sincere sympathy in their great loss.

E. P. F.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Microme Co., 727 Atlantic Ave., Boston 11, Mass.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Middle Section: Cleveland, Ohio, Jan. 15, 1951.

Southern Section: Miami, Fla., Jan. 17, 1951.

Western Section: San Francisco, Calif., Jan. 27-28, 1951.

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Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, Pa.

Meeting: Havana, Cuba, 1952. For information, address Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

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Chairman of Section on Otolaryngology: Dr. Howard P. House.

Secretary of Section on Otolaryngology: Dr. Edwin Scobee.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.

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